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Table of Contents, Volume 69

MARCH, 1960—NUMBER 1

	PAGE
I—The Mechanism of Bone Conduction. George W. Allen, M.D. and César Fernandez, M.D., Chicago, Ill.	5
II—Internal Laryngocele Treated by Endoscopic Excision and Fulguration. R. Melvin Butler, M.D. and Willard F. Goff, M.D., Seattle, Wash.	29
III—The Effects of Abnormal Body Temperature upon the Ear: Cooling. W. L. Gulick and R. A. Cutt, Newark, Del.	35
IV—The Intratympanic Muscle Reflex as a Protective Mechanism Against Loud Impulsive Noise. David A. Hilding, M.D., Fort Knox, Ky.	51
V—Vestibular Lateropulsion. Nicholas Torok, M.D. and Alan Kahn, M.D., Chicago, Ill.	61
VI—Epithelial Neoplasms of the Larynx. A Departmental Review. James A. Maher, M.D., Ann Arbor, Mich.	73
VII—Cerebrospinal Otorrhea. Jose Ferrer, M.D. (by invitation), Chicago —	88
VIII—Experimental Observations on Postural Nystagmus. II. Lesions of the Nodulus. César Fernandez, M.D., Rene Alzate, M.D., John R. Lindsay, M.D., Chicago, Ill.	94
IX—Myoblastoma of the Larynx. Stephen F. Balshi, M.D., Philadelphia, Pa.	115
X—Auditory Discrimination and Visual Perception in Good and Poor Readers. C. P. Goetzinger, Ph.D., D. D. Dirks, M.S., and C. J. Baer, Ph.D., Kansas City, Kans.	121
XI—Variables Involved in Automatic Audiometry. Aubrey Epstein, Ph.D., Pittsburgh, Pa.	137
XII—Histogenesis of the Ampullary Cupola. Consuelo Savin V., M.S., México D.F.	142
Scientific Papers of the Annual Meeting of the Massachusetts Eye and Ear Infirmary Alumni Association	155

	PAGE
XIII—The Whispering Syndrome of Hysterical Dysphonia. Richard Thomas Barton, M.D., Beverly Hills, Calif.	156
XIV—Tracheotomy in Head and Chest Injuries. Burton D. Bryan, M.D., Fall River, Mass.	165
XV—Chronic Hyperplasia of the Mucous Membrane of the Pharynx. J. Charles Drooker, M.D., Boston, Mass.	170
XVI—The Temporal Bone in Paget's Disease. Louis E. Griffey, M.D., Natick, Mass.	178
XVII—Laryngectomy: Past, Present and Future. Norman Jesberg, M.D., Los Angeles, Calif.	184
XVIII—Psychogenic Hearing Loss in Children. A Preliminary Report. Robert E. Klotz, M.D., Albert W. Koch, E.D.D., Thomas P. Hackett, M.D., Boston, Mass.	199
XIX—Results of Nine Hundred and Thirty-Nine Stapes Mobilization Operations. C. M. Kos, M.D., J. L. Shapley, Ph.D., P. B. Iles, B.A., Iowa City, Ia.	206
XX—Some Unsolved Problems of Stapes Mobilization. Donald K. Lewis, M.D., Boston, Mass.	222
XXI—Psychological Considerations in the Management of Cancer. Daniel Miller, M.D., Boston, Mass.	236
XXII—Osteoma of the Frontal Sinus. William W. Montgomery, M.D., Boston, Mass.	245
XXIII—Laryngeal Granuloma After Tracheal Intubation. Julio Quevedo, M.D., Guatemala City, Guatemala	256
XXIV—Postnasal Discharge. Daniel J. Reagan, M.D., Worcester, Mass.	263
XXV—Reappraisal of Seventy-Five Cases of Radical Neck Dissection for Carcinoma of the Larynx. George F. Reed, M.D., James B. Snow, Jr., M.D., Boston, Mass.	271
XXVI—An IBM Record Card for Stapes Surgery. John J. Shea, Jr., M.D., Memphis, Tenn.	280
Abstracts of Current Articles	284
Books Received	293
Notices	297
Officers of the National and International Otolaryngological Societies	307

TABLE OF CONTENTS

v

JUNE, 1960—NUMBER 2

	PAGE
XXVII—Combining Antibiotic with Antigen Therapy in Cases of T.O.E. (Yeast and Fungus) Allergy. Ben T. Withers, M.D., Houston, Tex.	309
XXVIII—Stapedoplasty. Further Observations. Arthur L. Juers, M.D., Louisville, Ky.	316
XXIX—Granular Cell Myoblastoma of the Larynx with the Presentation of Two Cases. William L. Walter, M.D., Staten Island, N.Y.	328
XXX—An Unusual Case of Adenocystic Carcinoma of the Parotid Gland. William Lippy, M.D., Charles Giffin, M.D., William H. Saunders, M.D., Columbus, Ohio	340
XXXI—The Role of Trauma in the Pathogenesis of Temporomandibular Joint Arthrosis. Ekrem Gozum, M.D., Minot, N.D.	348
XXXII—Blood Loss During Adenoidectomy and Tonsillectomy Measured with Radioisotopes. R. L. Ruggles, M.D., Cleveland, Ohio.	360
XXXIII—The Structure of Nerve Elements in the Major Salivary Glands of the Human. Irving M. Blatt, M.D. and William G. Bunto, M.D., Ann Arbor, Mich.	375
Scientific Papers of the American Laryngological Association	
XXXIV—The Doctor's Responsibility to His Patient. William J. McNally, M.D., Montreal, Canada	387
XXXV—Physiology of Olfaction and Gustation. Lloyd M. Beidler, Talla- hassee, Fla.	398
XXXVI—Electron Microscopy of the Olfactory and Gustatory Pathways. A. J. de Lorenzo, M.D., Baltimore, Md.	410
XXXVII—Laryngeal Manifestations of Systemic Disease. Walter H. Malon- ey, M.D., Cleveland, Ohio	421
Scientific Papers of the American Otological Society	
XXXVIII—Early Embryology of the Auditory Ossicles and Associated Struc- tures in Relation to Certain Anomalies Observed Clinically. Barry J. Anson, Ph.D. (Med.Sc.), Jerome S. Hanson, M.S. and Shafik F. Rich- any, Ph.D., M.D., Chicago, Ill.	427
XXXIX—Components of Electrical Responses Recorded from the Cochlea. Nelson Yuan-sheng Kiang, Ph.D. (by invitation) and William Tower Peake, Sc.D. (by invitation), Cambridge, Mass.	448

	PAGE
XL—Human Cochlea Responses to Sound Stimuli. R. J. Ruben, M.D., J. E. Bordley, M.D., G. T. Nager, M.D., J. Sekula, M.D., G. G. Knickerbocker, M.S.E.E., U. Fisch, M.D., Baltimore, Md.	459
* XLI—Some Physiological Factors in Inner Ear Deafness. Merle Lawrence, Ph.D., Ann Arbor, Mich.	480
XLII—Distribution of Hearing Loss in Various Populations. Aram Glorig, M.D. and James Nixon, B.A., Los Angeles, Calif.	497
XLIII—Tissue Reaction Following Reconstruction of the Oval Window in Experimental Animals. Richard J. Bellucci, M.D. and Dorothy Wolff, Ph.D., New York, N.Y.	517
XLIV—The Management of Middle Ear Lesions Simulating Otosclerosis. Francis A. Scoy, M.D., San Francisco, Calif.	540
XLV—Vein Plug Stapedioplasty for Hearing Impairment Due to Otosclerosis. C. M. Kos, M.D., Iowa City, Ia.	559
XLVI—Partial Stapedectomy. J. V. D. Hough, M.D., Oklahoma City, Okla.	571
XLVII—Stapedectomy. Harold F. Schuknecht, M.D., T. Manford McGee, M.D., Detroit, Mich., and Bernard H. Colman, M.D., Edinburgh, Scotland	597
XLVIII—Acceleration as a Means of Determining the Sensitivity of the Components of the Non-Auditory Membranous Labyrinth. Walter H. Johnson, Ph.D., J. Brydon Smith, M.D. and Joseph A. Sullivan, M.D., Toronto, Canada	610
XLIX—Localization Difficulty in Monaurally Impaired Listeners. Richard Viehweg, M.A., Richard A. Campbell, M.S., San Francisco, Calif.	622
 Obituaries	
George Morrison Coates	635
Felix Robert Nager	640
Prof. Luigi Pietrantonio	641
 Officers of the National and International Otolaryngological Societies	642

SEPTEMBER, 1960—NUMBER 3

L—Vitamin A and Keratinization. Studies on the Hamster Cheekpouch. Donald J. Lawrence, A.B., Howard A. Bern, Ph.D., Monte G. Steadman, M.D., Berkeley, Calif., San Francisco, Calif.	645
LI—Some Aspects of the Biochemistry of Acoustic Trauma. Yasushi Koide, M.D., Masaru Konno, M.D., Yoshie Yoshikawa, M.D., Makoto Yoshida, M.D., Yuichi Nakano, M.D., Masao Nagaba, M.D., and Masanori Morimoto, M.D., Niigata-Shi, Japan	661

TABLE OF CONTENTS

vii

	PAGE
LII—Effects on Round Window Potentials of Localized Changes in Cochlear Temperature. Alfred H. Chambers, Ph.D., and George G. Lucchina, M.D., Burlington, Vt.	698
LIH—Etiology of Unilateral Total Deafness. Studied in a Series of Children and Young Adults. G. Everberg, Copenhagen, Denmark	711
LIV—Psychic Factors in Hearing Loss. Jane C. Farley, Ph.D., A. J. Derbyshire, Ph.D., David F. Austin, M.D., William W. Waldrop, M.S., Richard L. Carter, Ph.D., Clarence McCormick, B.A., and Peter J. Mills, M.S., Chicago, Ill.	731
LV—Vestibular Responses of Some Deaf and Aphasic Children. Benjamin Rosenblüt, M.D., Robert Goldstein, Ph.D., and William M. Landau, M.D., St. Louis, Mo.	747
LVI—Neurologic Observations on a Population of Deaf and Aphasic Children. Robert Goldstein, Ph.D., William M. Landau, M.D., Frank R. Kleffner, Ph.D., St. Louis, Mo.	756

Scientific Papers of the American Laryngological Association

LVII—Cinefluorography in the Pre- and Post-Operative Management of Laryngeal Cancer. John A. Kirchner, M.D., James H. Scatliff, M.D., Donald P. Shedd, M.D., New Haven, Conn.	768
LVIII—Phonation. Clinical Testing Versus Electromyography. David W. Brewer, M.D., F. Bertram Briess, New York, N. Y., and K. Faaborg-Andersen, M.D., Nykobing, Denmark	781
LIX—Posterior Choanal Atresia. Daniel C. Baker, Jr., M.D., Jules G. Waltner, M.D. (by invitation) and William Novick, M.D. (by invitation), New York, N. Y.	805
LX—Findings Referable to the Upper Part of the Respiratory Tract in Wegener's Granulomatosis. Henry A. Brown, M.D., Lewis B. Woolner, M.D., Rochester, Minn.	810
LXI—The Cerebral Cortex and Hearing. Lamar Roberts, M.D. (by invitation), Gainesville, Fla.	830
LXII—The Parotid Gland in Mikulicz Disease and Sjogren's Syndrome. William Garth Hemenway, M.D., Chicago, Ill.	849
LXIII—Arytenoidectomy in Children. Robert E. Priest, M.D., Harold S. Ulvestad, M.D., Frank Van de Water, M.D., Robert J. Richardson, M.D. (by invitation), Minneapolis, Minn.	869
LXIV—Early Invasion of the Thyroid Gland in Carcinoma of the Glottis. Ricardo Tapia Acuna, M.D., Mexico, D. F. Mexico	882
LXV—The Role of Radiation Therapy in Carcinoma of the Larynx. Joseph L. Goldman, M.D., and Sidney M. Silverstone, M.D., New York, N. Y.	890

	PAGE
LXVI—Extramedullary Plasmacytoma of the Upper Air Passages. Julius W. McCall, M.D., Carl H. Bailey, Jr., M.D., Cleveland, Ohio	906
 Scientific Papers of the American Otological Society	
LXVII—Inner Ear Pathology in Deafness Due to Mumps. J. R. Lindsay, F.R.C.S. (Ed.), and Paul H. Ward, M.D., Chicago, Ill.	918
LXVIII—The Action of Enzymes on Human Middle Ear Effusions. Carl F. Gessert, Ph.D., Elizabeth S. Baumann, B.A., Ben H. Senturia, M.D., St. Louis, Mo.	936
Notices	956

DECEMBER, 1960—NUMBER 4

LXIX—Lymphangioma of the Tonsil. Report of a Case with a Critical Review of the Literature. George I. Harrison, M.D. and Lewis A. Johnson, M.D., New York, N. Y.	961
LXX—The Effects of Anesthetics upon the Ear. II. Procaine Hydrochloride. W. E. Rahm, W. F. Strother, W. L. Gulick and J. F. Crump, Princeton, N. J.	969
LXXI—Otolaryngology and Comprehensive Care: A Glance into the Future. Frederick T. Hill, M.D., Waterville, Maine	976
LXXII—Melkerson's Syndrome. Caesar N. Abu-Jaudeh, M.D., Beirut, Lebanon	989
LXXIII—The Effects of Abnormal Body Temperature upon the Ear: Heating. R. A. Cutt and W. L. Gulick, Newark, Del.	997
LXXIV—The Loss of Counter-Rolling of the Eyes in Three Persons Presumably Without Functional Otolith Organs. Lieutenant Richard C. Woellner, MC, USNR and Captain Ashton Graybiel, MC, USN, Pensacola, Fla.	1006
LXXV—Carcinoma of the Larynx: Comparative Experience in the Management of 951 Limited Lesions. Brian F. McCabe, M.D. and John E. Magielski, M.D., Ann Arbor, Mich.	1013
LXXVI—Diagnostic Applications of Radiophosphorus for Malignant Neoplasms in Otorhinolaryngology. Ken Nagatani, M.D., Kyoto, Japan	1020
LXXVII—Ultrasound in Ear, Nose and Throat Diseases. W. D. Currier, M.D., Pasadena, Calif.	1030
LXXVIII—The Sternomastoid Muscle Approach to Parotid Gland Tumors. Robert C. Kratz, M.D., Newport, Ky.	1040

TABLE OF CONTENTS

ix

	PAGE
LXXIX—The Association of Nerve Deafness and Retinitis Pigmentosa. Interval Report. Francis H. McGovern, M.D., Danville, Va.	1044
LXXX—Sublingual Swellings. A New Approach. R. Badrawy, M.D., Cairo, Egypt	1054
LXXXI—Middle Ear Muscle Protection from the Acoustic Trauma of Loud Continuous Sound. An Electrophysiological Study in Cats. F. Blair Simons, M.D., Palo Alto, Calif.	1063
LXXXII—Technique of High-Speed Photography of the Larynx. Henry J. Rubin, M.D., Beverly Hills, Calif. and Maurice Le Cover, Los Angeles, Calif.	1072
LXXXIII—Biochemical Changes in the Inner Ear Induced by Insulin, in Relation to the Cochlear Microphonics. Yasushi Koide, M.D., Shigeo Tajima, M.D., Makoto Yoshida, M.D. and Masaru Konno, M.D., Niigata-shi, Japan	1083
LXXXIV—Vocal Rehabilitation in a Boy with Esophageal Voice. Clinical Note. Friedrich S. Brodnitz, M.D., New York, N. Y.	1098

Scientific Papers of the American Broncho-Esophagological Association

LXXXV—Toxicity of Bronchographic Contrast Media. An Experimental Investigation. Philip M. Johnson, M.D., Montclair, N. J.; Walter R. Benson, M.D., William H. Sprunt, III, M.D. and William A. Dunnagan, M.D. (by invitation), Chapel Hill, N. C.	1102
LXXXVI—Is Congenitally Short Esophagus Truly a Rare Clinical Entity? William A. Lell, M.D., Philadelphia, Pa.	1114
LXXXVII—Treatment of Esophageal Strictures. Daniel C. Baker, Jr., M.D. and George C. Hennig, M.D. (by invitation), New York, N. Y.	1127
LXXXVIII—Occult Bronchogenic Carcinoma. Case Report. John R. Hilger, M.D., C.M. (by invitation), St. Paul, Minn.	1131
LXXXIX—Microscopic Suspension Laryngoscopy. Anthony N. Scalco, M.D., William F. Shipman, M.D., Harold G. Tabb, M.D. (by invitation), New Orleans, La.	1134
XC—Esophageal Defects in Dermatomyositis. F. Edmund Donoghue, M.D., R. K. Winkelmann, M.D., Herman J. Moersch, M.D.	1139
XCI—Relationship of Pulmonary Function Testing to Bronchology. Albert H. Andrews, Jr., M.S., M.D., Helen Moore, B.A. (by invitation), Chicago, Ill.	1146
XCII—Permanent Tracheostomy in the Treatment of Pulmonary Insufficiency. Arthur Q. Penta, M.D., Schenectady, N. Y. and Edgar Mayer, M.D., New York City (by invitation)	1157

	PAGE
Scientific Papers of the American Otological Society	
XCIII—Labyrinthectomy. Terence Cawthorne, F.R.C.S., London, Eng.	1170
XCIV—Effect of Middle Ear Muscle Action on Certain Psycho-Physical Measurements. Scott N. Reger, Ph.D., Iowa City, Ia.	1179
XCV—The Lurking Latent Cholesteatoma. Victor Goodhill, M.D., Los Angeles, Calif.	1199
Scientific Papers of the American Laryngological Association	
XCVI—Some Clinical Aspects of Aphasia. Joe R. Brown, M.D., Rochester, Minn.	1214
XCVII—The Use of Regional Flaps in Head and Neck Surgery. John J. Conley, M.D., New York, N. Y.	1223
Abstracts of Current Articles	1235
Books Received	1246
Notices	1249
Officers of the National and International Otolaryngological Societies	1255
Index of Authors	1257
Index of Titles	1260

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

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I

THE MECHANISM OF BONE CONDUCTION

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AND

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The lateralization and increase in loudness of bone conducted sound produced by occlusion of the external meatus was one of the first bone conduction phenomena described. According to Bárány¹ it was first reported by Tortu² and Wheatstone³ in 1827. It has had a most profound effect on the development of the theory of bone conduction.

The theory of Mach⁴ (1864) aimed to explain this occlusion effect. Bárány mentioned this theory for historical interest only since many of its details are scientifically inaccurate in the light of modern knowledge. In a general form, however, it seems to have some merit according to our own experience. The theory stated that vibratory energy is transferred directly to the labyrinth from the vibrating head. Since the sound conducting apparatus is well adapted for transmitting sound energy from the air to the labyrinth, then according to the law of reciprocity, it is equally well adapted for transmitting energy in the opposite direction. Obturation of the meatus decreases this drain of energy from the labyrinth. This, in turn, increases the amount of energy available for stimulation, so the tone becomes louder. Mach⁴ also observed that compression of air in the external canal decreased the loudness of the sound.

From the Section of Otolaryngology of The University of Chicago.

This investigation was supported by funds provided under contract B-682 (C⁴) with U.S. Public Health Service.

Lucae⁵ criticized Mach's theory on the basis of this decrease in loudness caused by compression of the external canal air. He pointed out that this compression surely reduced the ability of the conduction mechanism to transmit sound and, according to Mach's theory, would be expected to increase bone conduction rather than reduce it.

Lucae⁵ in 1864 performed an experiment, a forerunner to many similar experiments committing what Bárány calls the "classical error in bone conduction theory." Lucae, using a human ear, cemented an indicator on the ossicular chain and the drum membrane. When the stem of a tuning fork was touched against the bone of the preparation, vibrations of the drum and ossicles could be recorded. Lucae concluded from this that the ossicular chain participates in bone conduction. This inference is not warranted.¹ If the entire temporal bone including ossicles and drum move as a unit, then no stimulation can occur as a result of ossicular chain movement and no sound can escape via the chain. It is not the movement of the ossicles relative to the external recording apparatus that is of significance but the relative movement between the ossicles and the cochlea.

Berthold⁶ in 1872 measured the alternating air pressure in the external canal with a microphonic flame when a bone conduction stimulus was applied. Many others since have done the same with electronic microphones. A pressure wave set up between the eardrum and a stationary microphone has no more significance than the movements demonstrated by Lucae.

Herzog and Krainz⁷ were the first to construct a theory showing clearly how stimulation by bone conduction might occur through a compressional mechanism. Mach did not seem to realize that the presence of kinetic energy in the labyrinth is not alone a sufficient stimulus, and his theory was incomplete from this point of view. Herzog and Krainz made it clear that an alternating pressure difference on the two sides of the basilar membrane is required. They postulated an alternate compression and rarefaction of the otic capsule producing a corresponding reduction and expansion of the volume of the cochlea. The labyrinthine fluids were considered incompressible. The fluids could move because of the pliability of the oval and round windows. The oval window is stiffer than the round window so a greater part of the fluid moves in the direction of the round window during the compression phase, thus displacing the basilar membrane. Herzog and Krainz realized that in addition to being passively displaced, the ossicular chain would participate in any rhythmic movement of the skull and might impart independent active impulses to the labyrinth. Thus the theory of Herzog and Krainz is the first satisfactory compressional

theory of bone conduction. Stimulation is considered the result of compression of the otic capsule but is reduced by the inevitable participation of the ossicular chain. It contains the germ of an inertial theory of bone conduction later to be developed by Bárány. In 1930 Krainz⁸ concluded that near the natural frequency of the ossicular chain, the oscillations of the chain may furnish the major part of the stimulation.

Békésy⁹ in 1932 made many important contributions to the theory of bone conduction. He showed that it is possible to produce complete cancellation of a bone conduction tone by an air conduction tone in the cochlea. Matched air conduction receivers are placed on the subject's ears. Each of these is supplied with amplitude and phase controls. By suitable adjustments of these controls a tone delivered by a bone conduction receiver could be completely cancelled out and silence obtained.

He also studied the mode of vibration of the skull, showing that up to 800 cps the skull can be considered to be vibrating as a whole. In 1948 he repeated this work¹⁰ and showed that there was a progressively increasing difference in phase of movement of the two sides of the head until at 1800 cps they moved in opposite directions. At this frequency one full wave length occupies one-half the circumference of the head and the velocity of sound can be calculated to be 540 meters per second.

Békésy⁹ also observed that while obturation of the cartilagenous external auditory meatus improves the bone conduction threshold, no improvement occurs when the bony meatus is obturated. For this reason he attributed the occlusion effect to the inertia of the mandible producing alternating compression and rarefaction of the external canal. Zwislocki¹¹ later found some improvement for low tones when the bony meatus is obturated and concluded that some compression of the bony canal by the mandibular condyle was possible.

Békésy pointed out the importance of the greater volume of fluid on the vestibular side of the basilar membrane in producing displacement of the basilar membrane according to the Herzog and Krainz⁷ theory.

In 1938 Bárány published his famous monograph on this subject. Of his numerous valuable findings and conclusions only a few can be mentioned here. Most important for our purpose he seemed to establish inertial bone conduction on a scientific basis.

Bárány¹ adapted Békésy's apparatus for demonstration of cancellation of bone and air conducted sounds to make measurements of

phase and amplitude. A human subject is seated with head fixed by biting a cast of the teeth which is rigidly supported. A masking noise is delivered into one ear. A bone conducted tone is delivered to various parts of the head by a bone conduction receiver especially designed and mounted to deliver a uniform, measurable intensity of sound to the skull regardless of the position of the receiver on the head. An air conducted tone of the same frequency is delivered to the opposite ear by a loudspeaker. The current to the loudspeaker can be varied by amplitude and phase controls. The intensity of the bone conduction tone is kept constant. The amplitude and phase of the air conducted tone are varied until the bone conducted tone is cancelled out and only the masking noise is heard. Bárány assumes that under these conditions the amplitude of the air tone at the cochlea is the same as the bone conducted tone, but their phases are opposite. If his assumption is correct, then the measurement of phase and amplitude of the air tone becomes a measure of the relative phase and amplitude of the bone conducted tone.

Bárány found that a significant sound pressure field is generated in the air surrounding the head by the skull vibrations induced by the bone conduction receiver. In order to measure the true bone conduction he subtracted the intensity-phase vector of this air sound from that of the total bone conduction. His major finding was that if the side of the head is stimulated by bone conduction, the phase of the sound tends to be about 180° different in the two ears. The intensities tend to be about equal. This is strong evidence that the dominant mode of stimulation under these circumstances is by inertia of the ossicles. Bárány performed almost all of his experiments with a tone of 435 cps. For this critical work a separate specially designed receiver would be required for work at each different frequency. At 435 cps phase differences which might be due to differences in arrival time of a compression wave are negligible.*

$$\lambda = \frac{v}{f} = \frac{540 \text{ m-sec}}{435 \text{ sec}} = 1.2 \text{ m}$$

$$1.2 \text{ m} = 360^\circ$$

$$.25 \text{ m} = 70^\circ$$

Bone conduction due to inertia of the ossicles would be expected to be of opposite phase but equal amplitude in the two ears, for if the

*Based on the commonly used sound velocity in bone of 3000 meters/sec (actually based on the velocity of sound in ivory), we calculate the wave length to be about 7 meters. Based on Békésy's¹⁰ value of 540 meter/sec we calculate the wave length to be about 1.2 meters. Taking a path of 25 cm around the head this could still account for only about 70° of phase difference.

head is translated to one side then the inertia of the ossicles would tend to push one stapes into its vestibule and pull the other stapes out. Bárány conceived that the ossicular chain is well balanced to reduce the amount of bone conducted noise. The center of gravity of the ossicles is very close to the axis of rotation. When a small weight is added to the umbo the center of gravity is shifted away, the moment of inertia is increased and inertial bone conduction is improved.

Also according to Bárány, compressional bone conduction should be of the same phase in both ears but might be of different intensity. He believed that both forms of bone conduction, inertial and compressional, interacted and that under some conditions one was dominant and under other conditions, the other was dominant. For example, with the receiver on the frontal bone, bone conduction is reduced compared to the mastoid route. In the frontal route the direction of translatory vibrations of the skull approaches the axis of suspension of the ossicles so that inertial bone conduction is minimized.

Bárány went further in trying to define the conditions under which one or the other of the two forms of bone conduction was dominant by studying conduction deafness with the previously described apparatus. One of his subjects had particularly good control of his eustachian tubes and was able to sustain a negative intratympanic pressure produced by the Toynbee maneuver and even to regulate its degree to a certain extent. In this way he could produce a controlled conduction hearing loss. It was found that when the air conduction threshold was decreased 20 db, the bone conduction threshold was decreased by 10 db; but that when air conduction was further decreased, bone conduction remained unchanged. Bárány concluded that under these higher degrees of conduction deafness, inertial bone conduction was eliminated. When the phase of the bone conduction under this condition was measured with the receiver on different parts of the head, he found that the phase still varied, although not as much as in the normal ear. This led Bárány to the inference that the compressional components are not of the simple variety conceived of by Herzog and Krainz.⁷

Thus Bárány points the way back to a possible interpretation of his experiments in terms of a purely compressional theory of bone conduction.

The theory of Langenbeck¹² also attempts to explain the clinical phenomena of bone conduction in terms of the interaction of different modes of bone conduction. He only attempts to explain the lateralization for low tones in the Weber test. He views the stimulation of

the cochlea at low tones as the resultant of two different inertial forces. One is produced by movements of the skull relative to the brain such that when the skull moves forward the brain lags behind and compresses the contents of the posterior cranial fossa. This compression is transmitted through the internal auditory meatus to the cochlear fluids. The other consists of a sound wave generated in the external canal by mandibular inertia and transmitted through the ossicular chain with a slight delay in phase (relative to the first component) to the cochlear fluids. With frontal or vertex stimulation the cerebral and mandibular components will be almost in opposite phase and tend to cancel each other out at the stapes footplate allowing, however, discharge of pressure across the basilar membrane. This relative immobility of the stapes under normal conditions explains why fixation of the stapes by otosclerosis produces only minor degrees of change in the bone conduction threshold. Also since otosclerotic fixation eliminates the mandibular component with its phase lag, the resultant phase would now be leading relative to the opposite normal ear. The abnormal ear effectively hears the sound sooner, so the Weber is lateralized to that ear. The phenomenon of the false-Bing test described by Fournier and Rainville¹³ is also explained by Langenbeck. The false-Bing test is positive when occlusion of the external canal of the normal or perceptively deafened ear produces an increase of loudness of a sound lateralized to the opposite conductively deafened ear. This phenomenon can be regularly elicited in unilateral conduction deafness. Langenbeck assumes that with the tuning fork on the frontal bone, occlusion of the ear canal does not change the relative phase relationships but simply increases the loudness. The increased loudness is contributed by the occluded ear and the lateralization is contributed by the conductively deafened ear.

Recently Kirikae¹⁴ has reported several further experimental observations of importance. The relative movement between the isolated temporal bone and the conductive apparatus was measured. A capacitative probe was mounted on the temporal bone, and the probe tip was set near the tympanic membrane. Attaching the probe to the temporal bone instead of the table or other fixed structure prevents the "classical error." The amplitude of this relative vibration was shown to remain flat from 100 to 300 cps, then rise gradually to a peak at 1000 cps. The vibration above 2000 cps could not be measured by this method. The phase relation between the vibrating temporal bone and the conductive apparatus was measured with two capacitative probes. The phase difference was found to be small at low frequencies (20° - 25° at 200 cps to 400 cps), large at high frequencies (about 140° at 1000 cps), and the curve crossed the 90° line at 700 to 800 cps. If the

tympanic membrane were loaded with a metal weight, the amplitude of the relative movement between the temporal bone and tympanic membrane was increased in the region of 100 to 400 cps with a peak around 200 to 300 cps. Little or no increase was seen above 400 cps. The same type of weight was used to load the tympanic membrane of human subjects and thresholds for bone and air conduction were recorded. Bone conduction thresholds were improved with a first peak at about 300 cps of 12 to 36 db and a second peak at about 900 cps averaging 14 db. Air conduction thresholds showed a region of about 10 db loss at 200 to 400 cps and a second region of about 8 db loss at 900 cps with only small irregular losses for higher tones. The effect of loading the tympanic membrane on the cochlea microphonics of the cat were also measured. In this case the first peak of improvement for bone conduction was less prominent than the second peak. Air conduction losses were irregular but usually less than 10 db. The relative movement between the temporal bone and the conduction mechanism was also measured in the cat with results similar to those in the human. It is clear that the second peak cannot be explained on the basis of inertia bone conduction, because after loading the eardrum, the amplitude of vibration has remained unchanged while the cochlear microphonics or audiometric thresholds were improved. Kirikae explains this peak by the classical Herzog and Krainz⁷ theory. Kirikae also showed that blocking of either the oval or round window would cause the bone conduction thresholds for cochlear microphonics to become impaired. If both were blocked the impairment was greater than if either alone were blocked. He showed how compression of the cochlea can occur in a plastic model, and he measured the mobility to static pressure of the oval and round windows. The oval window was found to be twenty times as stiff as the round window. Kirikae endorsed a dual theory of bone conduction similar to Bárány's in which inertial stimulation interacts with compressional stimulation. He thought it necessary to assume that stimulation occurred by ossicular inertia in order to explain the first peak of the drum loading effect. He also assumed that stimulation occurred by inertia of the inner ear fluids in order to explain the decrease in bone conduction sensitivity after blocking the oval window and to explain the improvement of bone conduction thresholds after fenestration in otosclerosis (i.e., disappearance of the Carhart notch). According to the original theory of Herzog and Krainz⁷ fixing the stapes should improve the bone conduction thresholds and fenestration should impair them. According to the inner ear fluid inertia hypothesis of Kirikae, the decreased mobility of fluid caused by fixation of the stapes would diminish the stimulation caused by inertia of the inner ear fluid and explain the commonly observed results.

Other possible mechanisms of bone conduction exist. Relative movements of the ossicles may occur due to compression of the tympanic cavity. Energy may be transmitted to the external canal from the skull vibrations and from there to the cochlea through the ossicular chain with, however, a transmission loss at every bone, air, or soft tissue boundary. These and other routes almost certainly exist. The important question is whether their interaction is of any importance. If the sound pressure delivered by one route always exceeds that delivered by another route by 10 db, then the second route may be considered insignificant in determining loudness regardless of phase.

DEFINITIONS

Before outlining our hypothesis it is first necessary to explicate the definitions of some commonly used terms with the realization that all definitions are arbitrary conventions.

The *conduction mechanism* will be taken to include the pinna and external canal with their contained air, the tympanic membrane, the three ossicles with their ligaments, the intra-aural muscles, and the walls and air of the tympanic cavity excluding the round window. The *transformer mechanism* means the tympanic membrane, ossicles with their ligaments and the intra-aural muscles; and the *inner ear* means the intralabyrinthine fluids and membranes and the round window membrane. We will be concerned with the inner ear mainly as a vibratory mechanism. An alternate transformer mechanism, which is concerned only with bone conduction, will be designated the *bone-fluid mechanism*. This transformer mechanism is somewhat more vaguely conceived than is desirable, but must be similar to the compressional mechanism described by Herzog and Krainz⁷ and elaborated by Békésy.⁹ It is believed that a more complicated force than a simple uniform compression from all sides occurs. Wever and Lawrence¹⁵ have cast doubt on the possibility that compressional stimulation of this simple type is physically possible. There seems to be little reason, however, for assuming that a vibrating tuning fork or bone conduction receiver applied to the head would produce a compression of the otic capsule acting uniformly from all sides. Various torsional, shearing, or compressional forces acting more from one side than the other seem much more likely.

HYPOTHESIS

We postulate that when a vibratory force is transmitted to the skull from a bone conduction receiver, an alternating pressure differ-

ence is set up across the basilar membrane which results in movement of the basilar membrane as a consequence of direct action of the bone on the cochlear fluids. The pattern of displacement of the basilar membrane is independent of the points of introduction of the sound pressure.^{9,16,17} Transmission from bone to inner ear will be accomplished with a *transmission loss* and a *phase shift* at the bone-fluid boundary unless the impedance of the bone is equal to the impedance of the inner ear. Under other conditions energy will be reflected back into the bone. Sound pressure on the vestibular side of the basilar membrane may be reduced by passing out through the transformer mechanism. With a system of such complicated vibrational patterns as the drum-ossicular mechanism we do not assume that the principle of reciprocity will hold exactly, but it is likely that it would act as a step down transformer to more nearly match the impedance of the inner ear to that of the external canal. Perhaps its efficiency and resonance characteristics would be different when operating in a reverse direction.

Almost any condition affecting the conduction mechanism will do two things affecting bone conduction. First, it will tend to decrease the loss of sound pressure from the vestibular side of the basilar membrane by decreasing the impedance matching efficiency of the step down transformer. This tends to improve the bone conduction threshold. Second, it will increase the impedance of the inner ear and thereby increase the transmission loss at the bone-fluid mechanism. This tends to impair the bone conduction threshold. Furthermore, since the stiffness of the inner ear will be increased predominantly, a phase shift in the direction of leading occurs. Few conditions can be imagined that increase the mass of the inner ear. It might be supposed that loading or blocking the round window would increase the mass of the inner ear as we have defined it, but Wever¹⁸ has shown this to produce a greater air conduction loss for low than high tones. This shows it to be a stiffness effect.

An illustration will be given to show how the impedance of the inner ear may be changed. The figures given are probably quite inexact because differences in resonances of the different vibratory systems are ignored and impedance is taken to be equal to specific acoustic resistance. Wever, Lawrence, and Smith¹⁹ showed an average transmission loss of 28 db for air conducted sounds produced by removal of the tympanic membrane, malleus and incus when the sound is delivered directly to the oval window. 28 db loss corresponds approximately to a transmission coefficient of .001. The transmission coefficient is given by the formula:

$$T = \frac{4r}{(r+1)^2}, \text{ where}$$

T = fraction of energy transmitted

$$r = \frac{R_2}{R_1}$$

R_2 = the specific acoustic resistance of the second medium

R_1 = the specific acoustic resistance of the first medium

Substituting 41.5, the specific acoustic resistance of air, for R_1 and .001 for T we get a value which is reasonably close to 161,000, the specific acoustic resistance of sea water. Wever¹⁸ has shown that heavy blocking of the round window may produce an additional loss of 20 db or more above that produced by removal of the ossicular chain and drum. This further loss must come from an increase of acoustic resistance of the inner ear. Again applying the formula for the transmission coefficient, we find that the acoustic resistance of the inner ear has been increased to 1.6×10^7 . To show how this might affect bone conduction consider this further calculation. If the specific acoustic resistance of bone is taken to be 580,000 (Kobrak²⁰), then the transmission loss for sound passing from bone to inner ear will be found to be 0.67 or 1.7 db. If the round window is blocked then the transmission loss becomes .138 or 8.6 db. The difference or 6.9 db represents the transmission loss of energy from bone to cochlear fluid produced by this degree of round window block.

Fixation of the stapes would have a similar effect on the inner ear impedance but in addition would impair the action of the transformer mechanism. Again it should be emphasized that these figures only give the roughest approximations because of the vast oversimplification of these mechanisms.

EXPERIMENTAL PROCEDURE

The experiments were performed on three young adult men without significant hearing loss. Additional observations were made on two patients. All measurements on the three normal subjects were made with the Grason-Stadler model of the Békésy audiometer. Pure tones were pulsed. A Zenith Extended Range hearing aid receiver was used for determining bone conduction thresholds. A specially calibrated rubber cushioned PDR-10 receiver was used for measuring air conduction thresholds. White noise at 50 db or 60 db was delivered

to the opposite ear by a rubber cushioned PDR-10 receiver for masking.

All graphs show comparative measurements, the reference level being the normal threshold for the particular ear being tested. For bone conduction this means the ear with canal unoccluded; for air conduction the canal is occluded only by the receiver. It is important to remember that the ear being masked is occluded by the receiver in determining the proper level of masking noise since, as will be seen, occlusion of this type is very effective in improving the threshold for bone conduction.

Control thresholds were remeasured on the day of each test. A minimum of 3 tracings on the Békésy audiometer are superimposed and a graphic average taken. This becomes the zero decibel line against which the experimental threshold group of 3 tracings or more is compared. The graphs show the control curve subtracted from the experimental curve. Improvements in threshold are then recorded as negative numbers of decibels.

Three types of experimental manipulations were used: 1) Change in position of the bone conduction receiver; 2) loading the eardrum with a skin graft; and 3) occluding the external auditory meatus.

Skin grafts were used to load the tympanic membrane because they are easily handled, can be easily applied without adhesive, and are comfortable. They adhere well enough so that the head can be kept upright preventing the drum from being stretched by their weight. They are pliable and are expected to follow the vibrations of the drum. The grafts were cut three quarter thickness by a dermatome at surgery on man except for the heaviest graft which was full thickness. The pieces were trimmed to the desired size, weighed, and stored in aqueous merthiolate solution in a refrigerator. Three sizes were used: graft 1, 13.1 mg; graft 2, 34.1 mg; and graft 3, 114.2 mg. The grafts are applied to the eardrum with a Hartman's forceps and massaged lightly with a cotton-tipped applicator to remove bubbles and excess moisture.

Canal occlusion was produced in two different ways: 1, by simply applying a rubber cushioned PDR-10 receiver and 2, by a Mine Safety Appliance earplug. This consists of light rubber and fits into the outer part of the cartilagenous meatus. A needle is inserted through the earplug to balance the pressure in the canal and then the needle is removed.

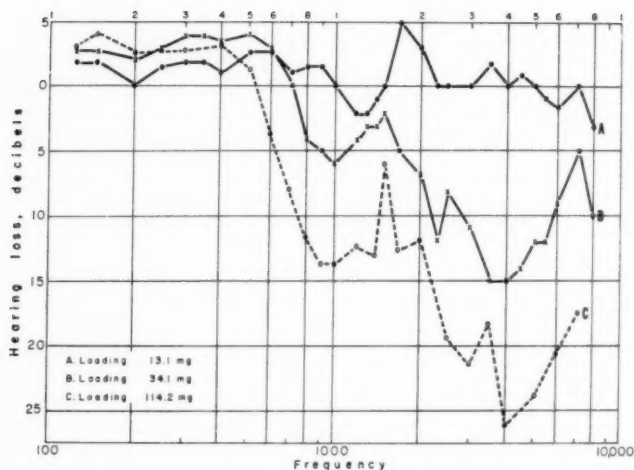


Fig. 1.—The effect, on the air conduction threshold, of loading the tympanic membrane with skin grafts of the different weights indicated. The general pattern is improvement for low tones and loss for high tones.

The steps of an illustrative experiment are given. First, a threshold curve is measured on the Békésy audiometer by superimposing 3 tracings, measuring for example, bone conduction on the left ear with the bone conduction receiver on the left mastoid and with 60 db white noise delivered to the right ear. Next, a skin graft is applied to the umbo of the left tympanic membrane and the new threshold measured in the same way. The first curve is subtracted from the second curve for chosen frequencies and plotted as shown in Figure 4. The result is a curve showing the threshold shift produced by loading the ear drum with a skin graft. Threshold shift is used as defined by Davis et al,²¹ i.e.: "is the deviation in decibels from an individual's own previously established reference audiogram." We shall use this term to indicate either improvement or impairment of threshold.

An attempt was made to make all measurements in a series without disturbing the position of the bone conduction receiver, but this is sometimes very difficult. It was found that with care to replace the bone conduction receiver in exactly the same place that curves could be reproduced with only slightly less accuracy than for air conduction. The graphically averaged curves will rarely deviate on repe-

tition more than 3 db for tones up to 3000, or more than 5 db for higher tones. This is true only for mastoid positions of the bone conduction receiver. When measuring from the frontal bone, a shift in the position of the receiver or a change in tension of the headband may produce a shift of 15 db for some frequencies. Even when the receiver is not deliberately moved, reproducibility of results is poor. Therefore the frontal bone measurements were excluded from this report.

The three normal subjects were sophisticated and practiced in the use of the audiometer.

RESULTS

EXPERIMENT 1. *Effect of Drum Loading on Air Conduction.* Air conduction thresholds were measured on subject No. 1 before and after applying the three different grafts. Figure 1 shows the threshold shift produced. All three curves show an improvement in threshold for low tones. Curve A crosses the 0 db line at 1000 cps, curve B crosses at 700 cps, and curve C crosses at about 530 cps. This shows a progressive shift in what we interpret to be the principal resonance toward the low frequencies. A second region of relatively high sensitivity is brought out in the 1500 cps region and does not seem to shift appreciably. Little if any impairment of threshold is seen for high tones with graft 1. A maximum loss of 15 db at 4000 cps is seen with graft 2 and a maximum loss of 26 db is seen at about 4000 cps with graft 3. This is about the weight of a full thickness graft that would be used for a tympanoplasty.

Comment: That these grafts do not act simply as low pass filters is shown by the actual improvement of threshold for low tones. These curves conform roughly in shape to the theoretical curve of Johansen²² for the effect of adding mass on the threshold curve. The fact that there is a peak of relatively increased sensitivity at 1500 cps in all 3 curves shows that the vibratory system of the ear cannot be accurately described as a system with only one net mass, one net stiffness, and one net frictional resistance. If this were the case a secondary resonance would be expected to bear a harmonic relation to the first and as the mass was increased, it should shift toward the low frequencies.

EXPERIMENT 2. *The False-Bing Test.* If a graft is placed on the left tympanic membrane, the Weber at 512 cps is lateralized to the left. If the left external canal is occluded, the Weber is also lateralized to the left. If the graft is placed on the left and at the same time the right canal is occluded, then the Weber is often lateralized to the left,

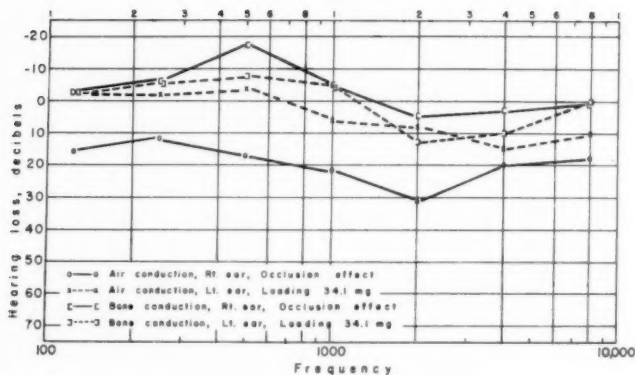


Fig. 2.—Thresholds for bone and air conduction from Békésy audiometer tracings in the form of a standard audiogram. The right external ear canal is occluded; the left tympanic membrane is loaded. In this case the Weber test showed lateralization to the left.

the side of the graft. The threshold curves under these circumstances are illustrated in the form of a standard audiogram (Fig. 2). No masking was used for air conduction, and 50 db white noise was used for bone conduction. The thresholds for low tones by bone conduction are improved in both ears. The maximum improvement is at 500 cps and is greater for the canal occlusion than for the drum loading. Both curves show some loss for high tones. The air conduction on the side of the graft is slightly improved for low tones and is impaired as much as 13 db for high tones. The air conduction with canal occluded is impaired from 12 db at 250 cps to 31 db at 2000 cps. The lateralization seems to depend rather critically on the size and placement of the graft and on the particular technique of occlusion. The larger grafts and deeper occlusions bring on this effect more reliably.

Comment: For this particular case the Weber lateralized to the left. This is an experimental false-Bing test and emphasizes the importance of phase in determining lateralization of the Weber. In terms of relative loudness the Weber should go to the right because, 1) that is the side of the better bone conduction, and 2) that is the side of the greater bone-air gap.

That this does not occur the same way with all combinations of grafts and canal occlusion is not surprising in view of the similarity

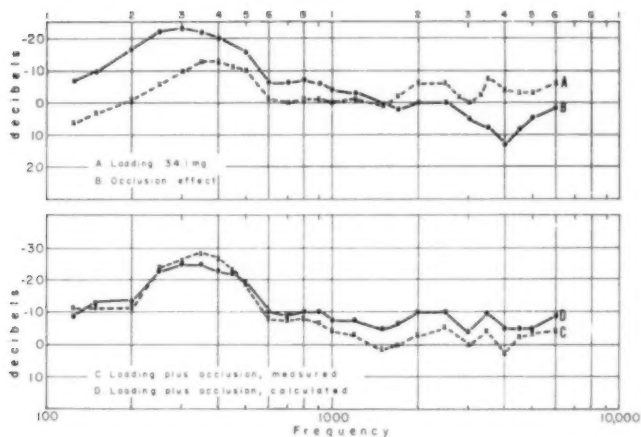


Fig. 3.—The effect of loading the tympanic membrane (curve A), occluding the external canal (curve B), and performing both simultaneously (curve C) on the bone conduction thresholds. No masking used. Curve D is derived from curves A and B in a manner described in the text.

of the nature of the two effects. This will be shown in our later discussion.

EXPERIMENT 3. *Effect of Drum Loading and Canal Occlusion on the Bone Conduction Thresholds.* In this set of experiments the left ear was tested with the receiver on the left mastoid. Curve A of Figure 3 shows the effect of graft number 2 on bone conduction. Curve B shows the effect of occluding the ear canal with a Mine Safety Appliance ear plug. Curve C shows the threshold shift when both canal occlusion and drum loading are performed simultaneously. No masking was used in any of these determinations. All three curves have a similar form except that canal occlusion alone shows a loss at one region, maximum at 4000 cps. The maximum improvement is greatest for the combined effect, curve C, 28 db; next highest for the occlusion effect alone, curve B, 23 db; and lowest for the drum loading effect alone, curve A, 13 db. Assuming that the interaural attenuation for bone conduction is 0, one would not expect to detect losses of sensitivity unless masking is used. It seemed desirable to perform the test without masking as well as with masking in order to test possible effects of the masking noise on the observed response.

Figure 4 shows the effect of the same conditions using 60 db white noise masking in the right ear. Again the maxima vary somewhat in position but are of the same general magnitude as when no masking is used. In all cases some loss for high tones is seen.

Comment: The graft effect is explained according to our hypothesis in the following way. Loading the eardrum decreases the efficiency of the step down transformer for most frequencies. This has two effects. First, at most or all frequencies there is increased immobility or impedance of the inner ear. This tends to impair the bone conduction threshold by causing increased reflection of energy from the bone-fluid mechanism. Second, the decreased efficiency of the transformer decreases the loss of energy from the vestibular compartment and tends to improve the threshold.

The loss of energy from the vestibular compartment would be maximal under normal conditions near the resonance frequency of the transformer. This can be predicted according to the transmission factor of Metz.²³

$$u = \frac{4\bar{w}}{(\bar{w} + 1)^2 + \bar{q}^2}, \text{ where}$$

u = fraction of energy transmitted

\bar{w} = acoustic resistance of the vibrating system
divided by the specific acoustic resistance of air

\bar{q} = reactance of the vibrating system divided by
the specific acoustic resistance of air

In this case we invoke the reciprocity principle at least in a semi-quantitative way. At the resonance frequency there is no reactance and \bar{q}^2 disappears from our formula. Transmission is at peak efficiency. The further we go from the resonance frequency the greater becomes the reactance and the less efficient is the loss of energy from the vestibular compartment. If not much is lost in this way to begin with, not much can be conserved by interfering with the transformer.

A similar explanation would hold for the occlusion effect except in this case the impedance match is disturbed not by an alteration of the transformer but by an increase of impedance of the external canal air.

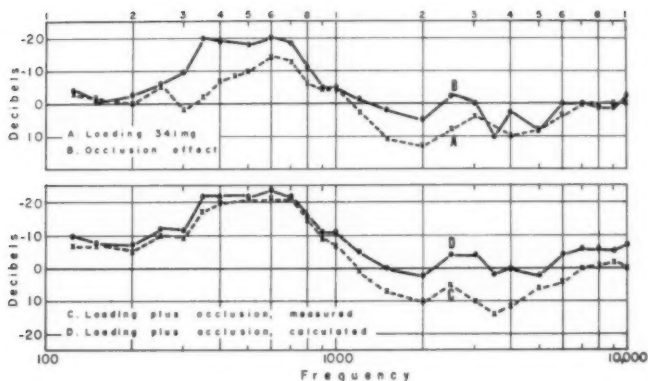


Fig. 4.—The effect of loading the tympanic membrane (curve A), occluding the external canal (curve B), and performing both simultaneously (curve C) on the bone conduction thresholds. The experimental procedure differs from that of Figure 3 in that 60 db white noise was used for masking. The text describes the manner of calculating curve D.

If the resonance frequency of our transformer is not changed too much, we would expect the effects of the drum loading and canal occlusion to be additive in terms of sound pressure conservation.

The D curves of Figures 3 and 4 illustrate this. The calculated curve is derived by taking the thresholds in db for the graft effect and the occlusion effect, converting them to sound pressure levels (positive db are used since sound is conserved) adding them and reconvertng to decibels. It is seen that the measured curves conform to the shape of the calculated curve much more closely than either of the parent curves. It would not necessarily be expected that the calculated and measured curves coincide exactly because the calculation does not take into account the losses at the bone-fluid mechanism. This experiment was repeated three more times in three different subjects with the same result.

EXPERIMENT 4. *The Effect of Unilateral Absence of the Mandible on Bone Conduction.* Two patients with unilateral absence of the ramus of the mandible were examined. In one, a hemimandibulectomy had been performed for carcinoma of the tonsil. Due to his illness it was not possible to obtain reliable audiograms. There was recurrent carcinoma around the left ear but the external canal, drum, and middle ear appeared normal. The Weber test with 512 cps tuning

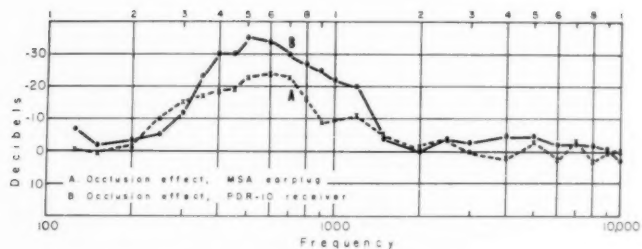


Fig. 5.—The effect, on the bone conduction thresholds, of occluding the ear canal with a Mine Safety Appliance earplug (curve A) and with a dead rubber-cushioned PDR-10 receiver (curve B). The latter produces a greater improvement.

fork showed no lateralization initially. When either canal was occluded the tone was heard to the side of the occluded ear and seemed to increase in loudness.

The other patient was a young man with clinically normal hearing. He had lost the ramus and condyle of his mandible as a result of childhood osteomyelitis. This was confirmed by x-ray. Again the Weber test showed 1) no lateralization initially, 2) lateralization to the occluded ear equally on testing either side, and 3) increase in loudness on occlusion of either ear canal which was equal on both sides.

Comment: Langenbeck's theory¹² can now be criticized. First, according to Langenbeck's theory, the occlusion effect should not occur in the absence of the mandible. Second, since the theory states that the mandibular component arrives at the cochlea later than the cranial component we can say that there is a relative phase lag in the transformer mechanism. We know that adding mass to the transformer would increase this phase lag. Then according to Langenbeck's theory, if one drum is loaded, the Weber test should show lateralization to the unloaded side whereas the lateralization is actually to the side of the graft. Third, absence of the mandible should eliminate the mandibular component on that side. The phase of the remaining cranial component would then lead the net phase of the other ear. The lateralization with no graft and no occlusion should be to the side of the missing mandible, but actually there was no lateralization.

EXPERIMENT 5. Comparison of the Occlusion Effects Produced by Ear Plug and by Earphone. Attention is next turned to an expla-

nation of Békésy's⁹ observation that obturation of the bony meatus does not improve bone conduction.

Bone conduction thresholds for the occluded left ear are plotted in Figure 5. 60 db white noise masking of the right ear was used. The occlusion effect produced by a needled Mine Safety Appliance earplug is represented in curve A. The occlusion effect of a dead rubber cushioned PDR-10 receiver is shown by curve B. It is seen that the receiver produced a much greater occlusion effect, the two maxima being 35 db and 24 db. The peak sensitivity occurs at about the same frequency in both curves.

Comment: If Békésy's explanation⁹ of mandibular stimulation were correct, then for an equal amplitude of mandibular compression a greater increase in pressure should occur in the smaller cavity. Of course, even the tragus moves when the mandible moves so the effective radiating sound surface is reduced by the Mine Safety Appliance earplug, but this should be more than compensated by the vastly reduced volume of entrapped air.

The hypothesis proposed here attempts to explain this effect by assuming that the obturation of the bony meatus produces such a big increase of impedance of the air entrapped in the canal that reflection of energy from the bone-fluid mechanism counteracts the tendency for sound to be conserved in the vestibular compartment. Within limits the more distal from the drum that the occlusion is produced, the less will be the loss at the bone-fluid boundary.

EXPERIMENT 6. *The Drum Loading Effect as Measured from Opposite Sides. Additional Hypothesis.* Next an attempt will be made to test Bárány's explanation¹ of the drum loading effect. If the ossicles are well balanced to reduce bone conduction because of inertia of the ossicles and this balance is then disturbed by loading the umbo, then inertia bone conduction would be increased. A consequence of this is that the higher the frequency, the greater would be the relative increase of stimulation produced by this inertial mechanism (the interested reader is referred to Bárány, chapter 12, for a detailed discussion of the magnitude of inertial bone conduction as a function of frequency). Yet the curve for the drum loading effect usually reaches a plateau at around 500 cps. This is a frequency at which the skull is translating as a unit. Then the only remaining thing that could cause the curve of the graft effect to turn down again as the frequency increases is that inertial bone conduction is cancelled out by compressional bone conduction arriving in opposite phase. According to

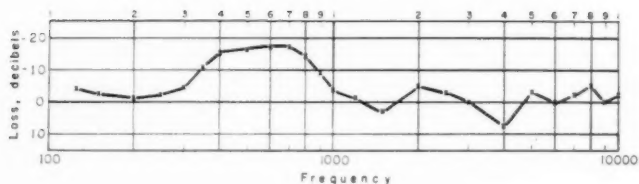


Fig. 6.—The effect, on the bone conduction threshold of the left ear, of loading the tympanic membrane with graft No. 2 (34.1 mg) when the receiver is on the right mastoid. 60 db white noise is delivered to the right ear for masking. The curve is of the same form as when measurements are made from the left mastoid (curve A of Fig. 4) but of slightly greater magnitude.

Bárány inertial bone conduction is in opposite phase but of equal loudness in the two ears, but pure compressional bone conduction is of the same phase but may differ in loudness in the two ears. Therefore, if compressional bone conduction counteracts inertial bone conduction in one ear, it would be expected to reenforce it in the other ear. Consequently if we measure the drum loading effect on the left ear from the right mastoid we should get a rather radically different result from that when we measure the drum loading effect on the left ear from the left mastoid.

Procedure: Sixty db white noise was delivered to the right ear. The bone conduction threshold was determined with mastoid receiver on the right, care being taken that it did not touch the masking receiver. The graft was then added to the left tympanic membrane and the new threshold determined.

Results: Figure 6 shows that the magnitude of the effect is slightly greater than that in Figure 4 but the form of the curves is very much the same. Peak increased sensitivity of both is about 600 cps and both return to the 0 db line at about 1200 cps.

Comment: To interpret this effect in terms of a dual theory of bone conduction in which compressional stimulation interacts with inertial stimulation, we must assume that the difference in phase between the two components is about the same in both ears.

This removes the major objection to a purely compressional theory; that is, Bárány's reasoning that opposition of phase indicates inertial stimulation.

It can be seen that our hypothesis makes it unnecessary to postulate stimulation by inertia of the inner ear fluids as does Kirikae¹⁴ in order to explain the decrease in bone conduction sensitivity after blocking the oval or round window or to explain the improvement in bone conduction threshold after fenestration for otosclerosis. In our own experiments we never observed the "first peak" described by Kirikae for drum loading. Kirikae's curves for air conduction show that his technique of drum loading is quite different from ours. His air conduction curves are not at all what would be expected from the pure addition of mass. It is probable that the stiffness of the tympanic membrane was increased by stretching. We do not believe that, with the information at hand, it is necessary to make a separate assumption to account for his "first peak" because, according to our hypothesis, there is not a simple relationship between the amplitude of the movement of the ossicular chain and the amount of improvement in the bone conduction threshold caused by drum loading. We would expect on other theoretical grounds, however, that the relative increase of amplitude of the movement between temporal bone and ossicles would be greater as the frequency was increased if the ossicular inertia were producing this increase of amplitude.

FINAL REMARKS

An attempt has been made to explain the effects on bone conduction of occluding the external canal (Bing test) and of loading the tympanic membrane in terms of a purely compressional stimulation. Also an explanation has been given for the lateralization of the Weber test and for the false-Bing test.

Important clinical phenomena that should also be explained are the Gellé test and Carhart's notch.²⁴

The Gellé test is performed by increasing the air pressure in the external meatus and measuring the threshold shift of bone conduction. If the ossicular chain is mobile there is a loss of threshold; if the chain is fixed the threshold is unchanged. There is little unanimity of opinion about the results of the Gellé test because of the difficulty of increasing pressure in the meatus without obturating it and without introducing adventitious noises. It seems fairly certain, however, that with a normal ossicular mechanism, there is an impaired threshold which is greater for low tones than for high tones. According to our hypothesis this would be explained in the following way. Pressure in the external meatus increases the impedance of the air in the external canal. It also presses the stapes footplate inward and stretches the

round window membrane outward. The pressure with which it does this is magnified by the transformer mechanism. Since the stiffness of both windows is increased the overall impedance at the bone-fluid mechanism is increased even more than if the oval window alone is affected. Since this is predominantly a stiffness increase, low tones are affected more than high tones. Since the impedance of both the external canal and the inner ear are affected somewhat proportionally, there is still some escape of sound through the transformer mechanism. If the canal is obturated as well, some of this loss could be prevented. Onchi²⁵ found that the Gellé effect could be reversed by obturation of the external canal.

The Carhart notch²⁴ is explained in exactly the same way as the drum loading experiment. In fact a Carhart notch is quite regularly found with drum loading (Fig. 4). Both drum loading and stapes fixation interfere with the transformer mechanism. Stapes fixation does so more profoundly so that transmission at the bone-fluid mechanism is more impaired. A slight increase in low tones was shown by Carhart²⁴ as part of his phenomenon. Stapes fixation as produced experimentally by Smith²⁶ has an effect on the bone conduction threshold similar to the Gellé test. This is done by tying a needle to the stapes. The needle is fixed in the eustachian tube. This probably stretches the round window membrane. Air conduction losses show that a large part of the effect is due to an increase in stiffness because low tones are more affected.

CLINICAL IMPLICATIONS

Although the bone conduction threshold is affected by any lesion of the conduction mechanism, except in rather exceptional conditions, it would seem to be a fairly stable measure to nerve function. Such subtle changes that prevent escape of energy from the cochlea without much affecting the passage of sound from the bone to the cochlea are probably not seen very often clinically, assuming that cerumen is removed from the canal before testing. Conditions affecting the round window might produce more profound losses of bone conduction.

When our patients make responses to the Weber test that we do not expect, we are less likely to attribute them to a preconceived idea on the patient's part. Lateralization often occurs to a fenestrated ear although the bone-air gap has been decreased so that it is less than that of the opposite untreated ear. This can now be understood.

Lateralization has questionable value in determining which ear is being tested by bone conduction. We must decide on other grounds whether the level of masking is adequate.

Our rules for the Weber test must be modified. In unilateral conduction deafness, lateralization will consistently be to the affected ear. If an asymmetrical perceptive deafness exists as well, the conduction deafness will be the more powerful determinant of lateralization. In bilateral conduction deafness when the lesions are of similar nature and site, lateralization will be to the side of the greater loss or will not occur. In bilateral conduction deafness where the lesions are of a different nature, the lateralization cannot be predicted from the air and bone pure tone thresholds.

SUMMARY

A hypothesis of bone conduction is developed, and an attempt is made to explain all of the important clinical and experimental phenomena of bone conduction in terms of this hypothesis. These phenomena have often seemed contradictory in terms of earlier theories.

The hypothesis proposed here states that while inertial forms of bone conduction may exist, the only important mode of stimulation is compressional.

Although all pathological states of the conduction mechanism affect bone conduction, it is a fairly stable index of nerve function except under unusual circumstances. Alteration of the conduction mechanism causes an alteration of the inner ear vibratory mechanism which changes the intensity and phase of vibrations delivered to the cochlea. It is this change of phase which primarily determines lateralization of the Weber. Also, changes in the conduction mechanism can prevent the escape of energy from the vestibular compartment of the cochlea through the ossicular chain. This improves the bone conduction threshold mainly around the resonance frequency of the conduction mechanism. The shape of the bone conduction threshold curves for occlusion of the external canal and loading the eardrum are explained.

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II

INTERNAL LARYNGOCELE TREATED BY ENDOSCOPIC EXCISION AND FULGURATION

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In the past 20 years many authors have discussed laryngoceles as to types, history, etiology, methods of removal, and possible complications. At present, there are still less than 100 cases of laryngoceles reported. We wish to review the literature on internal laryngoceles and present a case treated satisfactorily by laryngoscopic approach.

A laryngocele is a cystic dilatation of the saccule or appendix of the ventricle of Morgagni, with classification determined by extent of development into internal, external or combined types.^{1,2} An external laryngocele extends through the thyrohyoid membrane with cystic dilatation in the superficial layers of the lateral neck. The internal laryngocele remains medial to the thyrohyoid membrane. When both components are present, the laryngocele is classified as a combined type. Butler³ presents clear diagrammatic coronal sections of the larynx showing external and internal laryngoceles as well as the normal infantile and adult saccule.

The symptoms of a laryngocele as summarized by Horowitz⁴ include: 1) peculiar timbre, 2) alteration of voice tone, 3) hoarseness or weakness, 4) aphonia, 5) difficult respirations, 6) coughing bouts, 7) various degrees of dysphagia, and 8) lateral neck mass.

Unfortunately, late diagnosis is usually the rule rather than the exception. Indirect laryngoscopy is all that is needed to visualize the internal laryngocele adequately. A lateral neck mass which enlarges

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with increased endolaryngeal pressure (as in straining) is suggestive of an external laryngocele. However, coronal laminograms or tomograms of the neck are most helpful in confirming the diagnosis and in determining the extent of the laryngocele. Differential diagnosis includes ranula, dermoid cyst, cystic hygroma, branchial cyst, lipoma, aberrant thyroglossal cyst, and salivary glands obstructed with calculus or venectasia.⁴⁻⁶

Herberts⁷ presented Lawson's indications for operation as follows: a) in an internal laryngocele with symptoms of constriction or hoarseness; b) when there is difficulty in reducing the size of an external laryngocele by means of pressure, indicating presence of a valve mechanism; c) in infection; d) when working capacity is reduced (decrease in work tolerance).

Fatal asphyxia has been reported in at least two cases of infected laryngoceles.^{8,9} In both cases, tracheostomy or laryngostomy was performed too late. Boies¹⁰ reported a case of an internal laryngocele associated with a vocal cord papilloma and dysphonia plicae ventricularis. According to Jackson,¹¹ Schall reported a case of laryngocele associated with carcinoma of the larynx; and Leborgne, a radiologist, feels that the incidence of laryngocele in association with carcinoma of the larynx may be as high as ten to fifteen per cent.

A lateral neck approach, with variations, is the common method of total excision of external and combined laryngoceles;^{2,12-15} however, the method of removal of an internal laryngocele is considerably more controversial. O'Keefe¹ and Herberts⁷ advocate a lateral neck approach to an internal laryngocele, with O'Keefe using cotton-tipped applicators for dissecting the sac free.

Kucera and Priest¹⁴ reported a case of internal laryngocele removed by thyrotomy after a tracheostomy. Boies¹⁰ performed a laryngofissure for removal of a left vocal cord papilloma and a right internal laryngocele. He later noted recurrent right ventricular enlargement and clipped and cauterized the right ventricular band by direct laryngoscopy.

Jackson¹¹ states that in many cases of internal laryngocele, biting out some of the sac wall endoscopically is indicated. However, Keim and Livingstone¹⁶ state that attempts at destruction of the sac by cautery or partial excision through the laryngoscope are foredoomed to failure because of the impossibility of eradicating all of the lining membrane.



Fig. 1.—Tomogram - internal laryngocele (left) (2/19/59).

Walls² agrees that attempting to remove a laryngocele by cauterization or through a laryngoscope is to be condemned, as is x-ray therapy.

Holinger, Johnston and Schiller,^{17,18} in a series of 15 laryngoceles treated seven by endoscopic means (i.e., incision and drainage).

REPORT OF A CASE

J. M., a 59-year-old retired white male pipe fitter, was bronchoscoped in a tuberculosis sanatorium five years prior to the present admission, and was found to have a cystic mass projecting from the left lateral laryngeal wall just superior to the vocal cords. He was informed of its presence and faithfully visited an ear, nose and throat clinic every six months for observation. No great change in size was



Fig. 2.—Tomogram following endoscopic partial excision and fulguration (3/26/59).

ever noted in this mass. However, the patient repeatedly complained of hoarseness and a feeling of something which he wanted to swallow. His past history included a right nephrectomy, thyroidectomy for goiter, pulmonary tuberculosis and mild diabetes.

In February, 1959, he was admitted to the King County Hospital, Seattle, where, under general anesthesia, with a small cuffed endotracheal tube, the cyst was visualized using a Davis mouth gag. The cyst was incised, and there was a quiet pop with expression of air and subsequent collapse of the sac. The diagnosis was changed to left laryngocele, internal type. The surgery was concluded after noting that the sac had completely refilled. Several days later cervical laminograms revealed a left internal laryngocele measuring 2.5 x 4 cm which extended 1 cm across the midline.

Surgery was again performed in early March, 1959, with general anesthesia and small cuffed endotracheal tube. The laryngocele, which obscured the posterior two-thirds of the left cord, was well visualized with an adult Jackson laryngoscope held in place by a Lewy self-retaining bar. The superior and medial portions of the laryngocele sac were carefully excised with a long scalpel and Mathews forceps, and the margins fulgurated by diathermy electro-coagulation. At the conclusion of the procedure, the sac was not visible and only a few fragments of sac wall remained. The pathologist reported the tissue compatible with a laryngocele. Postoperatively, the patient did extremely well and was discharged three days later.

Cervical laminograms one month postoperatively revealed complete disappearance of the laryngocele with normal appearance of the true and false cords and ventricles. An eleven month follow-up confirmed absence of the laryngocele on mirror laryngoscopy. Also, the voice was improved.

COMMENT

Endoscopic partial excision with fulguration of an internal laryngocele may be adequate surgery to eliminate all symptoms and regain relatively normal anatomy.

In the poor risk patient, this simple procedure would be the therapy of choice.

The association of laryngeal cancer with laryngoceles, although infrequent, makes mandatory a careful examination and biopsy of each laryngocele.

SUMMARY

The authors have presented a case of internal laryngocele partially excised and fulgurated by endoscopic technique with excellent results. A review of the surgical approaches to the internal laryngocele is briefly presented, as well as symptoms, diagnosis, surgical indications, and complications.

1215 FOURTH AVE.

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III

THE EFFECTS OF ABNORMAL BODY TEMPERATURE UPON THE EAR: COOLING

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According to the electrical theory of auditory nerve stimulation, the small electrical potential generated by mechanical deformation of the hair cells of the organ of Corti triggers the impulses in the auditory nerve. Auditory nerve fibers form a complex dendritic network within the Deiters cells and thus are at the immediate site of the potential changes.

A reduction of the magnitude of the cochlear response results in a corresponding reduction of auditory nerve stimulation. Because hearing involves a complex chain of events and processes following cochlear stimulation, it may not be concluded that the cochlear response is sufficient to hearing. Nevertheless, any conditions which reduce the magnitude of the response must necessarily produce deleterious effects upon hearing.

Békésy³ has calculated that the mechanical energy of sound cannot account for all of the electrical energy of the cochlear potential. This suggests that the generation of the cochlear response is based upon metabolic processes. There is evidence that this is so.^{1,5,9,10} Accordingly, changes in the rate of metabolism produced as a consequence of abnormal body temperature might be shown to alter the magnitude of the cochlear potential, and thereby influence hearing. With the advent of reduced body temperatures in certain instances of heart surgery, it seems important to know whether or not reductions in body temperature could produce a temporary or permanent hearing loss. Furthermore, if a change in the cochlear potential does accompany alterations in body temperature, the nature of the change

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might provide valuable information for auditory theory in regard to the generation of the electrical activity of the inner ear.

In 1931 Adrian, Bronk, and Phillips¹ chilled the cochlear bone of a guinea pig with pieces of ice and found a weakening of the response. When the ice was removed, the cochlear response returned to normal provided that circulation had not been seriously affected. When circulation had ceased, however, the cooling depressed the response irreversibly.

Using a refinement of the technique of Adrian et al., Chambers and Lucchina⁴ pressed the tip of a copper wire cooled to approximately -40°C , to the cochlea of a cat. When the wire was placed at a point anteromedial to the round window, responses to tones of 500 and 1000 cps were reduced to 50 per cent of control values within six minutes. Responses to tones of 3000 and 4000 cps were reduced by less than 10 per cent of control values. In every instance, however, the response recovered fully within 20 minutes after the removal of the cold stimulus.

Both of the studies cited so far have been concerned with the effects of cochlear temperature on the magnitude of the cochlear response. Up to the present, very little attention has been given directly to the relationship between body temperature and the cochlear potential. There are, however, two studies which bear upon this relationship.

Wever and Bray⁸ noted that when a decerebrate cat was placed in an ice pack, no changes in the cochlear potential were observed until the cat died.

In 1950 Kahana, Rosenblith, and Galambos⁶ experimented upon the effect of reduced body temperature upon the cochlear potential in hamsters. By means of a copper coil through which ran hot and cold water, these experimenters changed the body temperature from 32°C (normal) to 18°C and then back to normal. The body temperature of each animal was recorded by means of a clinical thermometer placed in the cheek pouch on the side opposite the ear under study. Photographic records were kept of the cochlear response produced by clicks of 0.1 msec duration. The entire cooling and warming process took approximately three hours for each animal. Their results indicated that the latency of the response remained constant throughout the temperature range investigated. However, the amplitude of the response fell significantly with decreasing tem-

perature. The changes noted were reversible: when normal body temperature was restored, the cochlear response returned to its former level.

Data relating to the effects of temperature upon the cochlear response are contradictory. Cooling has been shown upon separate occasions either to decrease the magnitude of the response or to have no effect.

The purpose of the present experiment was to determine whether there exists a predictable and consistent relationship between subnormal body temperatures and the magnitude of the cochlear response. Recovery from temporary cooling was studied over extended periods of time in order to ascertain whether or not the response would recover. Heretofore this had not been done. It was hoped, too, that data obtained in this experiment would provide some information regarding the nature of the generation process of the cochlear response.

METHOD

Adult guinea pigs were employed as experimental animals. Body weights ranged from 336 to 700 grams. Their daily diet consisted of about 35 grams of rabbit chow (Purina) and a plentiful supply of fresh greens. Water was always available, and the average intake was 50 cc per day.

Anesthesia. Anesthesia was produced with ethyl carbamate (Urethane, Merck) in 20 per cent aqueous solution. The anesthetic was injected intraperitoneally in a dosage of 12.5 cc per kilogram of body weight. The dosage was sufficient to produce a surgical level of anesthesia within two hours and to maintain the preparation in good physiological condition for the duration of the experiment (up to 30 hours after injection).

In two animals the anesthetic caused respiration to become depressed. To prevent respiratory failure, 0.5 cc nikethamide (Coramine®) in 25 per cent aqueous solution was injected intraperitoneally.

Surgical Procedure. The tympanic bulla was approached laterally through a 1 cm incision in the skin immediately behind the pinna. Dissection was begun at a point 1 cm dorsal to the posterior tip of the mandible. This entry gave access to a part of the mastoid portion of the temporal bone anterior to the lambdoidal ridge. After removal

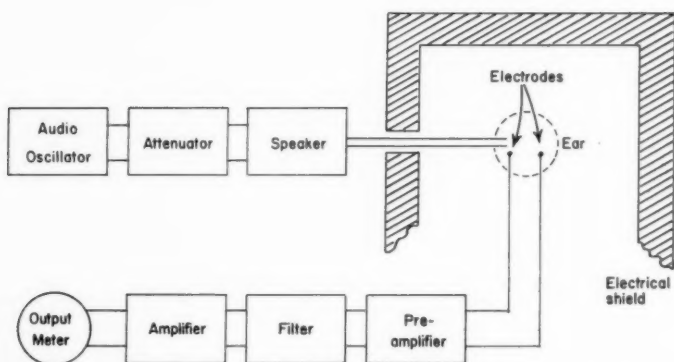


Fig. 1.—The arrangement of apparatus used to produce the stimulus and to measure the response.

of the periosteum, a hole (diameter 1 mm) was drilled with a small burr until the middle ear cavity was reached. The bone in this region is less than 0.5 mm thick. The proper place of drilling was midway between the lambdoidal ridge and the external auditory meatus. The opening was enlarged until the round window membrane was accessible for placement of a recording electrode.

Method of Stimulation and Recording. The stimulus was a pure tone (1000 cps) produced by an audio oscillator (Hewlett Packard, 200 AB). The signal from the oscillator fed into an attenuator (Hewlett Packard, 350 B) with a range of 110 db, variable in steps of 1 db. The output from the attenuator led directly to a 5-inch cone loudspeaker housed in a soundproof chamber.

Aerial sound was conducted into the external meatus through a tube leading from the speaker. A cannula at one end of the tube was sealed within the external meatus so that the tip of the cannula was approximately 4 mm from the tympanic membrane.

The active electrode was a piece of platinum foil soldered to a fine nylon-coated copper wire. The inactive electrode, made from a steel hypodermic needle, was inserted into exposed tissue surrounding the incision.

The tip of the recording electrode was placed upon the niche of the round window by means of a micromanipulator. The cochlear

potentials picked up at the electrode were amplified, filtered, and measured with an audio frequency spectrometer (Brüel and Kjaer, 2109) operating as a selective voltmeter. During the experiment each animal was isolated in an electrically shielded room. Figure 1 illustrates the arrangement of apparatus used to produce the stimulus and to measure the cochlear response.

Method of Measuring and Changing Body Temperature. Body temperature of the guinea pig was measured with a laboratory thermometer placed under the side of the animal as he lay on the operating table. This method of measuring body temperature proved to be as accurate as an intraperitoneal placement since the animal rested upon an insulated pad, and the ease of placement made it considerably more satisfactory. Measurements taken in this manner were also compared with those taken at the cheek pouch and anus on each of two animals. The discrepancies never exceeded 0.4°C . The thermometer and the animal were affixed to the operating table in order to avoid shifting. The glass of the thermometer stem was shielded from the heat and cold applied to the animal. Latency in measuring changes in body temperature undoubtedly occurred, since the changes had to permeate through the animal from the surface to which heat or cold was applied. However, since the changes were accomplished slowly, a near constant latency of one or two minutes was not considered to be critical in this experiment.

The animal was cooled with a plastic bag containing cold water (about 3°C) which was supported directly over the animal by three rods. So that respiration would not be hampered and a slow rate of cooling would be assured, the bag did not touch the animal. The skin area which was cooled extended from the lower thoracic region in a posterior direction and included the hind quarters.

The return of body temperature to normal was affected with a reflecting lamp located approximately 60 cm above the body. This lamp emitted heat sufficient to increase slowly the animal's body temperature to the desired level. The skin area affected was identical to the area already mentioned in connection with the cold stimulus.

PROCEDURE

The anesthetized animal was shaved with an electric clipper in the region of the head where the incision was to be made. After the bulla was reached, the bone was drilled to expose the round window. By enlarging the hole in the bulla, a large portion of the cochlea was

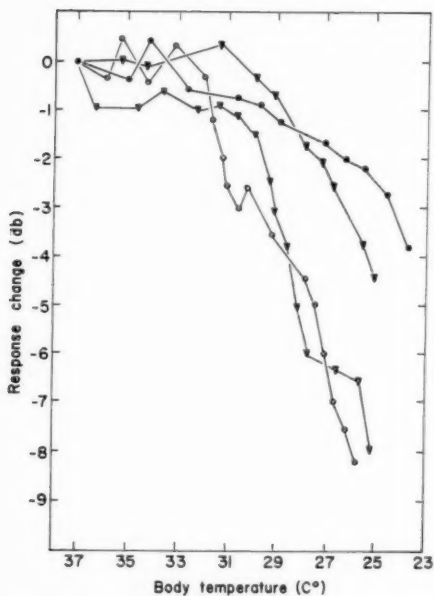


Fig. 2.—The effects of reduced body temperature upon the magnitude of the cochlear response to a 1000 cps tone for each of four guinea pigs. The changes in response are given in decibels relative to the output at normal body temperature ($37^{\circ}\text{C}.$). Rates of cooling for the data represented by circles and triangles were 4 and 6 degrees per hour, respectively.

exposed to the air of the shielded room. This was done in an attempt to hold the temperature of the cochlea constant regardless of body temperature. Air temperature at the bulla was measured with a second thermometer and was held constant at $26^{\circ}\text{C} \pm 1^{\circ}$. Thus, any substantial change in the cochlear response which accompanied changes in body temperature could not be assumed to be due to significant temperature changes within the cochlea itself.

To prevent the active electrode from changing position after it had been placed on the round window niche, it was necessary first to immobilize the head of the animal. This was done by tying the animal's snout around a rigid steel bar which ran across the surface of the tongue just posterior to the incisors. Thereafter, the sound tube cannula was inserted into the external meatus. A tight seal was effected by wrapping the pinna around the cannula and taping it

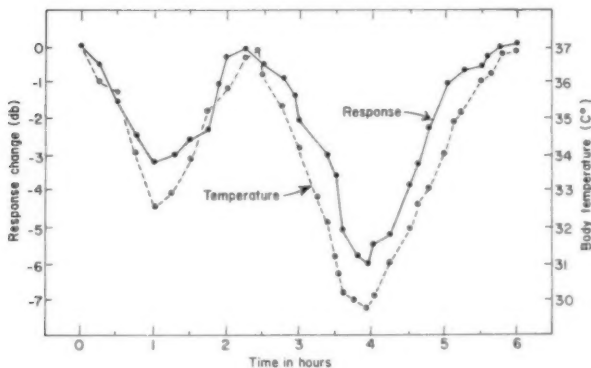


Fig. 3.—Curves showing the relationship between body temperature and loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate.

closed with plastic tape. Following the placement of the thermometers and electrodes, the stimulus was introduced to the ear. On three dead animals used to test for microphonics no artifacts were obtained.

Generally, it was necessary to raise body temperature to normal (37°C) since it tended to fall during experimental preparations. At normal body temperature the cochlear output was plotted as a function of the intensity of the stimulus (intensity function). The standard procedure was to start with an attenuator setting which produced no measurable response, and then to increase the intensity of the stimulus in steps of 5 db and plot the response in microvolts up to the point of overloading. After this reference intensity function was obtained, the attenuator was adjusted so that a response of 30 microvolts could be read at the output meter. The decibel setting at the attenuator was noted and thereafter referred to as the reference intensity, and this intensity was used during investigations of cooling and recovery.

During cooling and recovery, records of body temperature and the magnitude of the cochlear response were kept. Intermittently the respiration and pulse rates were noted and recorded. Changes in the magnitude of the response were measured in decibels relative to the output produced by the reference intensity at normal body tem-

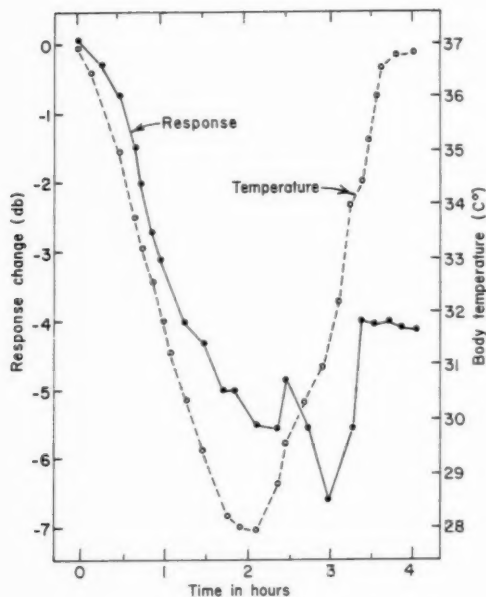


Fig. 4.—Curves showing the relationship between body temperature and loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate.

perature. At extreme temperatures a second intensity function was plotted for comparison with the one obtained initially.

Four animals were subjected to cooling until they expired, and four were used to investigate whether or not the effects of cooling were reversible.

An electrocardiograph was obtained on another animal, and changes in pulse rate were recorded continuously as body temperature decreased from normal to 25° C.

In a control animal body temperature was maintained at 37° C $\pm 1^\circ$ and the cochlear response was observed to be constant (± 1 db) for a period of 26 hours. The stability of the cochlear response

demonstrated by this animal agrees with data obtained in another study.⁷ Accordingly, changes which occurred under experimental conditions were assumed to be due to temperature variations in the body.

RESULTS

Data gathered in this experiment indicate that reductions in body temperature produce changes in the magnitude of the cochlear response. The curves presented in Figure 2 show the changes in the magnitude of the cochlear response which accompanied decreasing body temperatures. For the temperature change from 37 to 32° C the cochlear response showed only slight and unpredictable changes. However, with further reductions in body temperature each of these animals showed a loss in the response. Different rates of cooling were not responsible for the differences in the amount of loss among these preparations. The animals showing the least and greatest loss were cooled at the same rate (approximately 4° per hour), whereas the other two were cooled more rapidly (approximately 6° per hour).

Data gathered on four animals to determine whether or not the cochlear response would recover from the deleterious effects of lowered body temperature are presented in Figures 3, 4, 5, and 6. As shown in Figure 3, this animal demonstrated complete recovery after being cooled to temperatures of 32° C and 30° C. The two curves, one representing changes in the cochlear response and the other representing changes in body temperature, may be seen to parallel each other.

The results of more severe cooling are shown in Figure 4. Here the data presented show that when the body temperature of another preparation was reduced to 28° C, only partial recovery resulted when body temperature was returned to normal.

Data from a third animal used to study recovery from cooling are presented in Figure 5. This preparation was chilled to 26° C with a concomitant loss in the cochlear response of about 10 db. Raising body temperature back to normal had no immediate effect upon the magnitude of the response. The cochlear response showed a 10 db loss (± 1 db) for almost 3 hours after body temperature had returned to normal. Normal temperature (37° C) was maintained ($\pm 1^\circ$ C) until the animal expired 20 hours later. The response showed partial recovery (2.5 db) 10 hours after the experiment had begun, but during the next ten hours there was essentially no further recovery. At the time of death the response still showed a 6 db loss. Death produced a sudden and precipitous drop in the magnitude of the response.

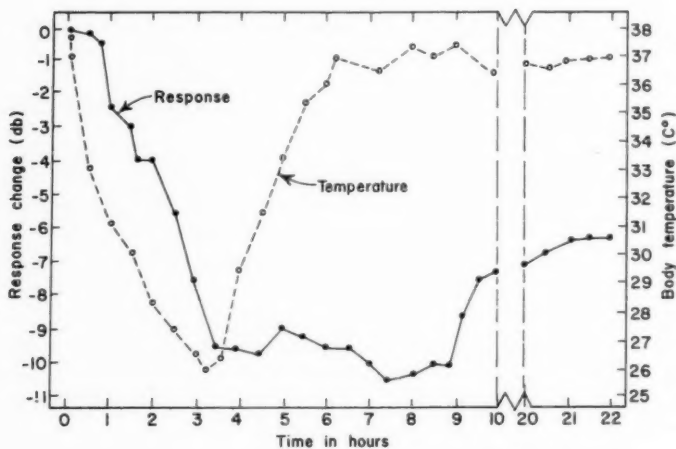


Fig. 5.—Curves showing the relationship between body temperature and loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate. During the 10 hours represented by the break in the abscissa the response remained stable at -7 db and body temperature remained constant at 37° C. ($\pm 1^{\circ}$).

The fourth animal on which recovery was studied underwent the most severe and rapid change in body temperature. Within 1.75 hours after the cold pack was applied to this animal, body temperature had fallen to about 24° C (a rate equal to about 8° per hour). During this time the response suffered a loss of about 4 db. Upon raising body temperature toward normal, the output of the cochlear response did not stabilize, nor did it show recovery. Instead, the response continued to decline until the animal expired. When death occurred, body temperature had risen to within 1.5° C of normal and the response had dropped about 10 db. These data are presented in Figure 6. The atypical behavior of this animal is believed to have been due to the rapid rate of cooling, a rate which may have interfered with essential life processes.

All animals remained in good physiological condition while data were being gathered. Respiration rate varied among the animals, but at all times it seemed sufficient to oxygenate arterial blood. Normal pulse rate for the guinea pig is 180. Reduction of body temperature

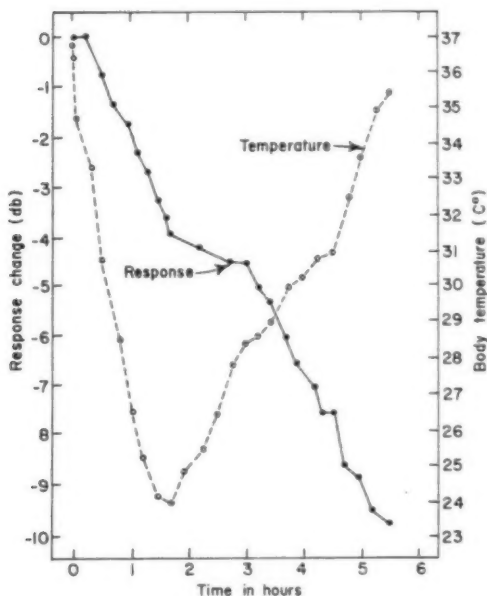


Fig. 6.—Curves showing the relationship between body temperature and loss in the cochlear response (in decibels relative to the output at normal body temperature) as a function of time. The response curve (solid line) is read against the left ordinate, and the temperature curve (dashed line) is read against the right ordinate.

to 25° C reduced the pulse rate to about 135, a rate which is not without the normal limits of fluctuation.

COMMENT

There are two possible explanations for the reduction in the magnitude of the cochlear response with changes in body temperature: one, that cooling the body slowed the metabolic processes responsible for the generation of the electrical response of the inner ear; and two, that cooling the body selectively damaged some of the hair cells of the organ of Corti. Let us consider each of these possibilities.

Metabolism. Present theory of the origin of the cochlear response states that it results from mechanical deformation of polarized hair cells of the organ of Corti. The theory assumes that the cell contents

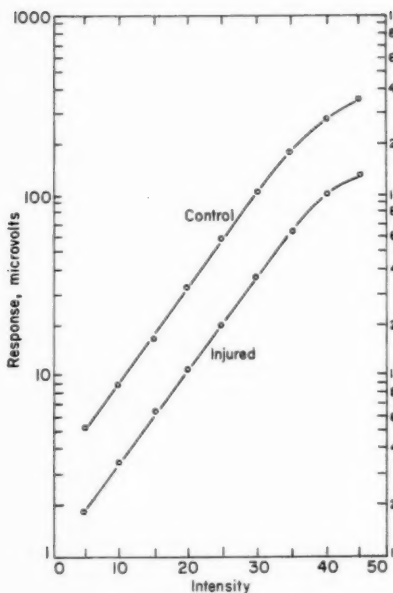


Fig. 7.—Intensity function plotted for a 1000 cps tone before and after injury by over-stimulation. Intensity values are expressed in decibels relative to .004 volts output at the oscillator. Injury was produced by a 4000 cps tone at 80 db sounded for three minutes.

and that the cell membrane provides a selective barrier to ions within and without the cell. Deformation of the hair cell changes the permeability of the cell membrane and allows an ionic flow.

Cochlear potentials vary in magnitude as a linear function of sound pressure up to the point of overloading. When overloading occurs, the function departs from linearity and the ear is endangered. At the present time there is only one tenable possibility that can account for the linear form of the intensity function over a range of intensities as extensive as 70 db; namely, that the voltage output of the individual hair cells bears a linear relation to the sound pressure and the number of hair cells in action does not increase with increases in intensity. To assume that the number of active hair cells increases with intensity, one must also assume that the output of the individual hair cell is decelerated to a degree sufficient to compensate exactly for the increased voltage contributed by the addition of hair cells.

That this combination of functions would hold true over a 70 db range is unthinkable. Furthermore, evidence that the function remains linear after many of the hair cells have been damaged makes this interpretation even more unreasonable.

Data from the present experiment indicate that changing body temperature produces a loss in the magnitude of the cochlear response. If a comparison is made between the loss in sensitivity produced by reducing body temperature and that produced by stimulation-deafness, it would seem that the loss in both instances is quite similar.

Intensity functions for an animal whose ear was experimentally injured by over-stimulation are shown in Figure 7. Both curves (control and injured) were plotted for a 1000 cycle tone. In Figure 8 are shown typical intensity functions plotted for a 1000 cycle tone at 37° C (normal) and 26° C. Upon examining these functions it can be seen that the changes that occurred between the intensity functions plotted at normal and reduced body temperature are similar to the changes that occurred between the control and injured functions plotted on a different animal. Sensitivity decreases and overloading occurs earlier. Nevertheless, the functions remain linear and their slopes do not change.

Because the slope of the functions obtained during sub-normal body temperature remained unaltered, it must be concluded that the active hair cells continued to operate in a linear fashion and at the same level of efficiency which was in effect prior to cooling. Whatever is the ratio of cochlear output to stimulus input, the same ratio must exist during and after cooling to effect the constant slope demonstrated by the data of this experiment. A general reduction in rate of metabolism is, therefore, an unsatisfactory explanation of the data of this experiment.

Cellular Injury. Since it is known that the loss in sensitivity produced by over-stimulation results from damage to a limited number of hair cells of the organ of Corti,² it can be inferred that similar injury occurred in the present experiment. It has already been pointed out that the rate of metabolism in the biological processes responsible for the cochlear potential could not have been changed in this experiment. The loss in sensitivity must therefore be due to a reduction in the number of active hair cells. Such a reduction would be reflected in a smaller voltage output for a given stimulus, and no change in slope would be expected. Accordingly, it is concluded that

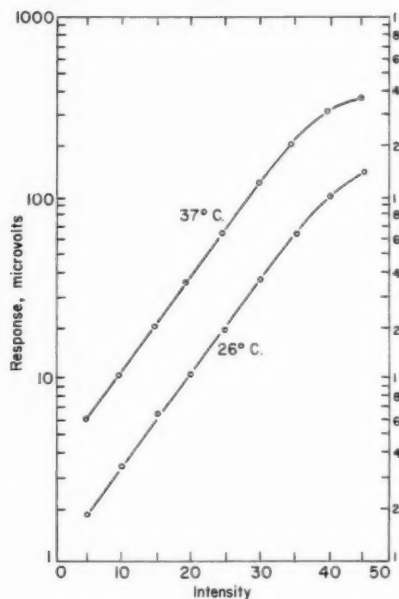


Fig. 8.—Intensity function plotted for a 1000 cps tone at 37° C. (normal) and 26° C. Intensity values are expressed in decibels relative to .004 volts output at the oscillator.

cellular damage to the hair cells of the inner ear is a satisfactory explanation of the effects of sub-normal body temperature upon the cochlear response.

What is required now is to consider ways in which reduced body temperature could injure the hair cells. The most direct way would be for the cold to influence directly the structure of the cell, either by altering its membrane structure or by producing excessive mechanical strain during its deformation under stimulation. In the Chambers and Lucchina⁴ study, wherein the cochlea of a cat was cooled locally with a probe of -40° C, the loss in the cochlear response to a 1000 cycle tone amounted to only 6 db. Since full recovery was noted within 20 minutes after the removal of the cold probe, their results strongly suggest that the structures of the cochlea are highly resistant to cold.

In the present study the changes in the cochlear temperature were probably very small. First, the head region generally underwent less severe temperature changes than the intraperitoneal temperatures would indicate because the head of the animal was shielded from the cold pack and remained in air of normal room temperature. Arterial blood probably took on heat from surrounding tissue as it circulated toward the head. Second, because only a small amount of blood circulates through the *stria vascularis* there is little opportunity for the transfer of heat from the endolymph bounding the hair cells to the blood in the *stria vascularis*. Third, the cochlear bone is extremely thin so that laboratory air circulating within the middle ear would tend to maintain the cochlea at room temperature.

If we conclude that the cochlea itself is highly resistant to temperature changes, and that, for the reasons indicated, the temperature changes of the cochlea in the present study were small, then we must look elsewhere for an explanation of the injurious effects of temporary reductions in body temperature upon the hair cells.

It is possible that injury to the hair cells was induced indirectly through changes in the chemical composition of the blood. However, the changes which occurred in the blood with cooling did not seem to influence the generation process of the cochlear potential. The hair cells which were active continued to behave in their characteristic manner without any loss of efficiency. We are left with the problem of determining what characteristics of blood chemistry could eliminate certain hair cells from the generation process without affecting the process itself.

SUMMARY AND CONCLUSIONS

Reductions in body temperature below normal were shown to reduce the magnitude of the cochlear potential, and thereby affect hearing. Four guinea pigs were chilled until they expired, and four others were used to study recovery.

On the basis of this study the following conclusions are offered:

1. Cooling body temperature below normal decreases the magnitude of the cochlear response, and the loss in response is positively accelerated with decreasing temperatures.
2. Upon returning body temperature to normal, the cochlear response shows full recovery provided that body temperature did not go below 30° C.

3. Only partial recovery occurs when body temperature is taken below 28° C, and the loss remains for prolonged periods (up to 20 hours).

4. The net loss in the cochlear response upon cooling is related to the decrease in body temperature in that the more extreme the cooling, the greater is the net loss observed in recovery periods.

5. Reductions of body temperature below 28° C (82.4° F) for periods as brief as 20 minutes may produce a relatively prolonged hearing loss.

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IV

THE INTRATYMPANIC MUSCLE REFLEX AS A PROTECTIVE MECHANISM AGAINST LOUD IMPULSIVE NOISE

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In 1959 Fletcher and Riopelle¹ were able to show that a short tone preceding each of a series of loud impulsive noises will provide protection against acoustic trauma. In order to demonstrate the value of the acoustic-reflex eliciting tone they measured the temporary threshold shift observed in human volunteers after exposing them to 100 rounds of blank 30 caliber machine-gun fire. They found that a tone of 100 cps at 98 db preceding the firing of each round for 200 milliseconds significantly reduced the temporary threshold shift.

They postulated that the tone provided protection by stimulating a reflex contraction of the muscles of the middle ear which introduced enough attenuation prior to the blast to lessen the harmfulness of the noise. It has been shown in animals that a contraction of these muscles can provide considerable attenuation of sounds, particularly low tones. Wiggers² measured the change in cochlear potential produced by the spontaneous contractions of the middle ear muscles which he observed under light dial-urethane anesthesia. He observed attenuation up to 45 db for 100 cps tones. Wever and Vernon³ measured cochlear potential changes produced by contralateral stimulation of the reflex and demonstrated attenuation of 20 db. Galambos and Rupert⁴ obtained similar values in awake cats with implanted round window electrodes.

Ordinarily, this attenuation is denied to the inner ear during loud impulsive noises, such as gunfire, because of the latent period of the intratympanic muscle reflex. The latency of the reflex has a duration of 10-20 milliseconds⁵⁻⁸ during which the energy of a shot could be transmitted to the inner ear. (The duration of the positive phase of a revolver shot is 2.7 milliseconds.⁹) Thus, it would be expected that the intratympanic muscle reflex is inoperative in protecting the ear from this type of noise, in spite of the results reported by Taruya.¹⁰

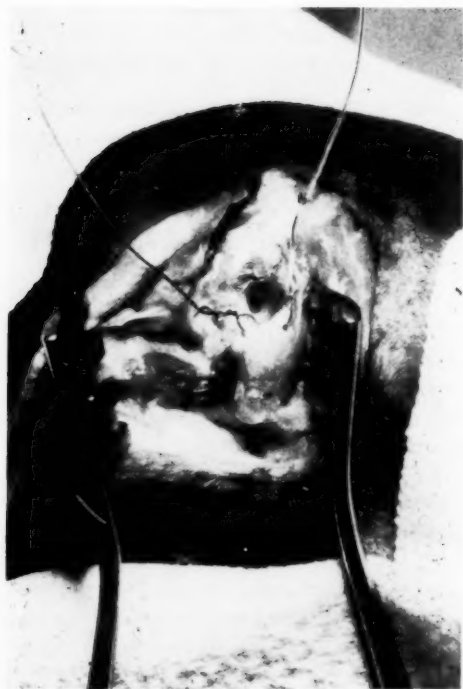


Fig. 1.—The bulla of the cat has been opened after separating the overlying soft tissues and inserting a self-retaining retractor. An opening into the bulla permits visualization of the round window membrane. The electrode has been laced through small perforations at the edge of the opening, and the ball-tipped end brought into gentle contact with the round window membrane. An indifferent electrode has been tied to the remaining perforation.

He exposed rabbits, with the tensor tympani stapedius tendon severed on one side, to the sound of cracker-ball explosions. There was more histological evidence of damage in the ears with cut tendons. However, the increased susceptibility to loud noise of these ears might be explained on the basis of alteration in the support of the ossicular chain produced when the tendon was severed.

The purpose of this paper is to present a preliminary report on experiments designed to test the value of Fletcher and Riopelle's "protective tone" against enough noise exposure to produce evidence of

permanent acoustic trauma in unprotected animals, and to determine if this effect is dependent on intact stapedius or tensor tympani tendons.

The extensive literature on the relationship of the intratympanic muscle reflex to protection has been discussed in detail elsewhere^{7,11,12} and will not be reviewed in this report.

METHODS

Preparation of the Animals. In order to obtain the data reported in this report, as well as for preliminary studies, 28 cats were implanted with round window electrodes according to the method used by Galambos and Rupert⁴ for recording cochlear potentials in awake cats.

Enamel-coated 0.005 inch (No. 36) stainless steel wire was used for the round window electrode. A ball-tip was formed on one end in a mercury-arc, and a PE 10 polyethylene tube was slipped over the wire. An indifferent electrode of No. 30 stainless steel wire was also fastened to the bulla.

Sodium pentobarbital 25mgm/kilo was injected intraperitoneally for anesthesia, and the head and neck shaved and scrubbed. The incision was made behind the ear extending onto the neck, and the soft tissue dissected to expose the bulla. A dental excavator was used to enter the bulla, and the opening was enlarged with a curette. Four small perforations were made around the perimeter of the opening. The electrode was laced through three of the perforations and the ball-tip placed in gentle contact with the round window (Figs. 1 and 2). The indifferent electrode was tied to the remaining perforation, the electrodes were brought through a small separate scalp wound, and the skin incision closed with 3-0 plain catgut. For the first five days after the operation, the animals were given oral tetracycline biotherapy.

The opposite ear was prepared in the same way, except that the tendon of the stapedius was cut or the attachment of the tensor tympani to the malleus was severed. In order to provide adequate visualization for this, the partition dividing the bulla into two compartments had to be partially removed.

Recording of the Cochlear Potential. After the few days required for the animals to recover from this procedure, the cochlear



Fig. 2.—A detailed view of the electrode tip in contact with the round window. The electrode consists of a No. 36 enamel coated stainless steel wire covered with 10 P E polyethylene tubing.

potential sensitivity was recorded. The animals were gently restrained in a leather bag, and a pure tone presented to them from an overhead speaker (University 8-inch speaker energized through a Fischer amplifier by a Maico audiometer). The potentials were picked up by the implanted electrodes and fed into a Grass AC preamplifier and cathode follower. The output of the preamplifier was fed into a Dumont 304A oscilloscope. A pure tone of 500, 1000, 2000, and 4000 cps were presented to the animals, and a record was made of the sound pressure level required to produce a cochlear potential of 10 V at each frequency.

Exposure to Noise. A 30 caliber machine gun was used as a source of loud impulsive-type noise. Blank rounds were fired with an



Fig. 3.—The 30 caliber machine gun was mounted outdoors. The cats were placed in a cage 50 cm above the muzzle of the gun and exposed to 2000 rounds. Half of the animals were exposed with a protective tone preceding each shot. This tone was presented from the four-inch speaker above the cage.

interval of 2-6 seconds between rounds. The gun was mounted outdoors and the animals were placed in a wire cage 50 cm above its muzzle (Fig. 3). As each round was fired, it is estimated that the cats were exposed to a peak noise of level of 155-160 db (Kryter, K: personal communication). After preliminary experiments, it was decided to use 2000 rounds fired from the gun as the standard noise exposure. The gun was fired by means of a solenoid which was energized through a delay circuit as described by Fletcher and Riopelle.¹ A pause of 200 milliseconds occurred between closing the switch and the detonation of the round. During this time, a tone of 1000 cps could be presented to the animals from a four-inch dynamic speaker suspended over their heads. (The tone was produced by a transistor type oscillator, and amplified so that the sound pressure level at the cat's head was 100 db).

Procedure. The animals were tested at daily intervals until three or more base-line recordings had been made. They were then separated into two groups. The first group consisted of five cats, and was exposed to 2000 rounds with each round being preceded by a short tone. The stapedius tendon had been divided in one ear of three of these animals, and the tensor tympani on one side severed in two. The

second group was made up of five cats, and was exposed to 2000 rounds without the protective tone. In this group, four cats had had the stapedius tendon cut on one side. Two of the animals in this group had active electrodes in only one ear prior to exposure.

Following exposure, the animals were tested at daily intervals for 20 days. After this period, the animals were again anesthetized with intraperitoneal sodium pentobarbital and were perfused with saline and formalin. The bullae were opened and examined. Histological studies will be performed after the temporal bones have been decalcified and sectioned.

RESULTS

Every ear exposed to 2000 rounds without the protective tone developed a marked loss in sensitivity. Of the four ears with intact muscle tendons in this group, two showed a response only at 4000 cps. The other intact ears showed no response at any frequency, even after 20 days.

In contrast, an average change of only 2 db was found in the ears with intact tendons exposed to 2000 rounds with the protective tone preceding each shot. These animals showed no significant alteration in cochlear potential sensitivity either immediately after the exposure or during the period of follow-up. However, the animals of this group with the stapedius tendon divided showed an average change of 25 db immediately following exposure, even though they also had the benefit of the protective tone. There was no tendency towards recovery observed.

The ears with cut tensor tympani tendons showed an average change of only 4 db after being exposed to 2000 rounds with the protective tone.

Until the histological studies have been made, the post mortem findings cannot be discussed in detail. However, on gross examination, it was striking how little reaction was visible in the bulla after this type of electrode implantation. In one ear of cat No. 24 and of cat No. 26 there was a small amount of polypoid tissue in the round window niche. There was no visible reaction to the electrodes in the other animals.

Severing the tensor tympani or stapedius tendon caused some adhesion formation. Usually, the severed stapedius tendon appeared

to be replaced by a thin band of scar tissue. In cat No. 23, there seemed to be a fracture dislocation of the head of the stapes. However, there was apparent preservation of the continuity of the ossicular chain.

The electrode had penetrated through the round window for a distance of 2 to 3 mm in the two ears where no microphonic response could be elicited prior to exposure.

The data from the preliminary experiments show that in five ears with intact tendons, exposed to 1000 rounds without the protective tone, the average immediate loss was 19 db, and 11 db two weeks later. At the same time, two ears with cut stapedius tendons were exposed to 1000 rounds, and developed an immediate loss of 29 db, with recovery to 18 db after two weeks. Five ears with intact muscle tendons were exposed to 2000 rounds with the protective tone and developed an average change of only 4 db. (Previously, these animals had been exposed to 500 rounds with little or no residual loss in sensitivity, but in order to prevent any confusion of the data, they are not included in the principal series.)

COMMENT

The data show much less change in sensitivity in the ears given a short tone preceding each blast than in those exposed without the tone. Not a single ear with intact muscle tendons showed a loss at any frequency of more than 17 db after being exposed to 2000 blank rounds with the protective tone, and the average change was only 2 db. On the other hand, virtually every ear exposed to the same number of rounds without the protective tone developed a loss of sensitivity of at least 30 db, with a complete loss of response even to 100 db stimulation at most frequencies.

Even though this experiment has been performed with a small number of animals, comparison of the data from the ears with intact tendons confirms the value of Fletcher and Riopelle's acoustic-reflex eliciting tone.

The ears with a severed stapedius tendon seemed to derive no significant protection from the tone. The animals with a normal middle ear on one side and a cut stapedius tendon on the other, after exposure to 2000 rounds with the protective tone, showed no change on the side with the intact intratympanic muscle tendons, whereas the ears with the cut stapedius tendons showed an average loss of 25 db.

A similar effect from severing the tensor tympani was not observed. However, the pre-exposure responses of these ears were not particularly sensitive. When these ears were examined at autopsy, the electrodes were resting against the bony margin of the round window, and were not in optimal position for picking up cochlear potentials. Galambos and Rupert⁴ found that the tensor tympani seems to be of less importance than the stapedius in protecting the ear against exposure to loud noise. They made use of cats implanted with chronic round window electrodes in which the tendon of the tensor tympani or stapedius was severed on one side. The animals were exposed to a 2000 cps tone at 85 db for three minutes. A temporary depression of cochlear potential response was observed after this exposure in the animals with the tendon of the stapedius cut. There was less change in the normal ears or those in which only the tendon of the tensor tympani had been cut.

More work is required before the findings discussed in this report can be regarded as conclusive. The histological appearance of the middle ears will be studied when the sections become available. Comparison of the cochlear sections should provide further data for evaluation of the protective value of the reflex. It is necessary to obtain more complete data on the effect of severing the tensor tympani tendon.

SUMMARY AND CONCLUSIONS

The value of a short tone preceding an impulsive noise in preventing damage to the inner ear has been evaluated in cats implanted with round window electrodes. Two groups of animals were exposed to 2000 blank rounds fired from a 30 caliber machine gun. For one group, each round was preceded by a tone of 1000 cps at 100 db, lasting 200 milliseconds. The other group was exposed without the tone. The tendon of the stapedius or tensor tympani was severed in one ear of most of the animals.

1. No loss in sensitivity of the cochlear responses was observed in the animals with intact stapedius tendons who were given the benefit of the protective tone.
2. The animals exposed without the protective tone developed a significant loss in sensitivity.
3. A significant loss in sensitivity occurred in the ears with severed stapedius tendons even when the protective tone was used. No

alteration in response occurred in the animals with severed tensor tympani tendons under the same conditions.

4. These data support the findings of Fletcher and Riopelle¹ that a short tone preceding an impulsive type noise reduces its harmfulness.

5. Since the protective effect is not present with a cut stapedius tendon, it is reasonable to assume that the protective tone acts by causing a stapedius muscle reflex, and the contraction of this muscle provides enough attenuation to protect the inner ear from this type of noise. However, more data must be collected in order to prove that this assumption is correct.

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V

VESTIBULAR LATEROPULSION

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The present discussion reporting experiences with a vestibular testing method not based upon nystagmus, is intended to be a preliminary one. Ever since the first reliable clinical vestibular testing was proposed and later popularized by Barany,¹ vestibular nystagmus became the yardstick in the evaluation of vestibular function. It was not arrived at arbitrarily, that such a clinical approach developed and ultimately won universal acceptance. Nystagmus is regarded as the most sensitive vestibular reaction. It is a sign which is relatively simple to recognize and is measured and recorded with considerable accuracy. The human test-subject, or patient, does not exhibit any other such recognizable reactions or signs when the vestibular apparatus is stimulated. On the other hand, the neuroanatomical pathways of the vestibulo-oculomotor reflex arch are more or less known and understood. Any physiological or artificial stimulation of the vestibular apparatus will generate neural impulses along this reflex arch, followed by the appearance of the nystagmus.

The central vestibular apparatus has several other relations and connections in addition to the nystagmus pathway, but, in the human subject, none of the vestibulosomatic reflexes are so apparent or readily observable. Consequently, all attention has been directed and concentrated upon the single phenomenon, termed "vestibular nystagmus." It is visible even through unaided observation. It is not, however, a simple phenomenon. The great variation of the nystagmic response in normal subjects needs a clearer explanation. It is often disappointing that very broad limits within a nystagmus reaction have to be considered as normal. The uncertain significance of various nystag-

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mus qualities such as the relationship between the fast and slow component, i.e., frequency and amplitude, is disturbing. Recent studies through the medium of nystagmography only serve to confirm the shortcomings in the interpretation of this type of vestibular reaction.

The necessity and effort to extend the scope of observation beyond the vestibular nystagmus is not a new one. Barany² carried out elaborate studies to test and evaluate what he termed the "past-pointing reaction" as a sign of normal or abnormal vestibular stimulation. Wodak and Fischer³ modified Barany's technique to the extent that instead of raising and dropping the arms, they observed the straight forward outstretched arms in a resting position at the height of the shoulders. It was called the "arm tonus reaction," which is as sensitive as the past-pointing maneuver and gives additional information relative to the function of the cerebellum. The Romberg test is so commonly employed that it does not require further elucidation.

All of these testing attempts are based upon the known neural connections existing between the vestibular and pyramidal systems (vestibulospinal tract) and integrated by the cerebellum. Any timing or other quantitative measurements of these reactions have been, however, uncertain and impractical. The sensitivity of available methods of testing is far beyond the sensitivity of the nystagmus reflex as observed through Frenzel's or Bartels' glasses or even by unaided observation. As a consequence of these facts, the general consensus has not favored giving too much time to these less practical observations.

Another approach designed to give information of vestibular sensitivity attempted to utilize the neural connections between the central vestibular and the autonomous nervous systems. Frenckner and Preber⁴ constructed an apparatus to measure the change in the resistance of the skin during vestibular stimulation. Such stimulation tends to increase parasympathetic tonus. Consequently, an increase in the activity of the sweat glands ensues. Sensitive and specially constructed electrodes are placed on the skin surface (forehead), which are able to indicate minute alterations in the moisture of the skin. By proper amplification, graphic recordings are produced. This method is one of the very few in recent years which attempts to bypass the absolute dependence upon vestibular nystagmus. However, recording the skin resistance during vestibular stimulation does not render the conventional nystagmus observation or recording dispensable. As Preber⁵ states, the test is very time-consuming; several repetitions are frequently needed but the examiner is enabled to detect



Fig. 1.—Electric rotating chair with photoelectric nystagmus and lateropulsion recording assembly.

increased sensitivity, e.g., in pilot candidates who are not aware, or unwilling, to admit such shortcomings.

For the past several years, all patients requiring an otoneurological examination have been tested with our nystagmographic equipment.^{6,8} Stimulation is applied through one medium, or several if needed: 1) rotation with an electrically driven rotating chair; 2) thermic stimulation, executed either in the sitting position with our standardized method of weak stimulation, or, if necessary, through the Fitzgerald-Hallpike technique in the supine position; or 3) postural stimulation.

All of these tests have been recorded by photoelectric nystagmography.⁶⁻⁸

Our electric rotating chair has a capacity of varying velocities from $22\frac{1}{2}$ to 180 degrees per second (Fig. 1). It was observed that,



Fig. 2.—Rotating chair equipped with lateropulsion recording device.

in certain instances, particularly when applying faster speeds, the patient's trunk deviated visibly in one direction at the start of the rotation, and in the other direction after the chair was suddenly stopped. This is not an unexpected nor unknown reaction, but evoked an interest for a better analysis of what is known as "lateropulsion."

Lateropulsion is closely related to other vestibulomuscular responses such as past-pointing, the arm tonus reaction, etc. Detailed studies of pioneer vestibular investigators Hogyes and Ewald⁹⁻¹¹ demonstrated that the vestibular apparatus has a constant influence upon the body musculature and a balanced tonus of the muscles is dependent upon normal vestibular function. A stimulated vestibular apparatus increases the tonus in certain muscle groups, with a decrease in the antagonistic muscles, and vice versa. Magnus and DeKleyn,¹² as a result of extensive animal experimentation, described various vestibulomuscular reflexes and in man even attempted to utilize certain vestib-



Fig. 3.—Test subject in rotating chair for simultaneous nystagmus and lateropulsion recording. 1. Vertical metal deflection bar; 2. Strain gauges attached to the metal bar; 3. Belt connected to bars and applied around chest of subject; 4. Photoelectric nystagmograph, eyepiece; 5. Cable of pulsometer; 6. Recorder-cable connectors. Note the tilted back rest and the resting of the subject's arms.

ular neck reflexes as signs of vestibular sensitivity. Lack of technical equipment applicable to human subjects, however, prevented any exact measurement of this vestibular reaction. Attempts were recently made by Japanese investigators, Utsumi et al,^{13,14} to demonstrate the vestibular tonus variations through electromyography.

It was felt that a graphic recording of the movement of the body, or more specifically of the per- and postrotatory lateropulsion would offer more enlightenment in the interpretation of this reaction. As long as this would be carried out simultaneously with the already existing means of recording nystagmography, it was possible to obtain a dual measurement of the postrotatory reactions. Therefore, an apparatus for the measurement and recording of the lateropulsion was constructed. The instrument is made up of four basic components:

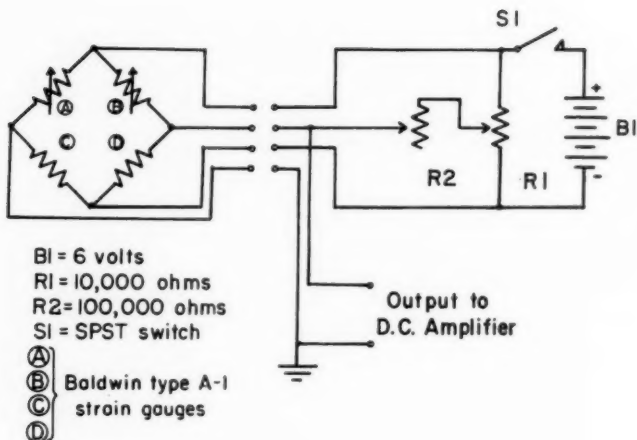


Fig. 4.—Schematic diagram of pulsometer circuit.

the first is the sensing element, represented by two flexible metal strips mounted vertically on either side of the subject. Each metal bar is fastened securely at its lower end to the rotating chair, and to the chest of the subject through a belt arrangement at the upper end (Fig. 2). Thus, as long as each metal strip is connected under tension, any motion of the subject's chest is reflected in a change in the shape of the metal strip. The second component is the transducer element, consisting of two sensitive strain gauges (Baldwin Type A-1), one mounted on the external surface of each of the metal strips, two inches above its point of attachment to the chair (Fig. 3). These strain gauges are connected into an electronic bridge circuit in such a manner that a deflection of the vertically mounted metal strips results in a proportional electrical signal (Fig. 4). The output signal of the bridge circuit is then applied to the third component, a low level direct current amplifier. This must be of the chopper type (Offner Type 190), as other types of DC amplifiers tend to drift excessively when amplifying such small signals. The output of this amplifier is then fed into a direct writing oscillograph unit which further amplifies these signals, and records them as a vertical graph plotted with time as the abscissa. The gain of the total amplifying system is adjusted so that a lateral deflection of the chest of one millimeter results in a vertical deviation of ten millimeters of the recording pen.

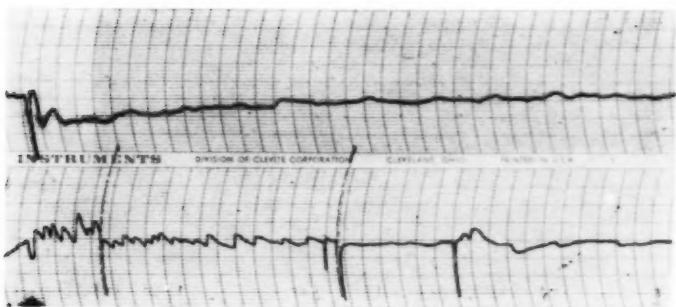


Fig. 5.—Simultaneous tracing of postrotatory lateropulsion and nystagmus. Counterclockwise rotation. Speed $45^\circ/\text{sec}$. Abrupt stop of the chair marked with arrow. The graphs are calibrated. For the pulsogram (upper graph) the distance between two sharp horizontal lines represents 0.5 mm body deflection. For the nystagmogram (lower graph) the same distance equals 5° deviation of the iris. The speed of the paper is set to 1 sec. between two vertical bars. Total nystagmus duration is 16 sec. The recovery of lateropulsion 29 sec. The maximum deflection of the body 0.8 mm.

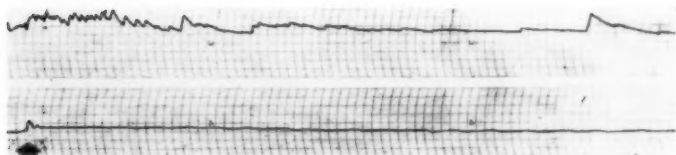


Fig. 6.—Clockwise rotation. Slightly prolonged postrotatory nystagmus. The lateropulsion record shows a small inertia wave (at the chair stop). The total deflection is 0.2 mm, the recovery time 39 sec.

All possible limitations of the free movement of the body must be eliminated. Heavy or bulky clothing is removed. The light, half-inch wide belt fastened to the metal bars is tightly applied around the chest of the patient. The back of the chair is deflected backward or entirely removed so that the patient is without support to his back. The arms and hands should not rest on arm rests or be held on any part of the chair. The arms are bent forward without support and the hands are held in a supine position, resting on the thighs (Fig. 3). Finally, the test subjects are instructed not to sit stiffly but comfortably and relaxed in the chair. The subjects selected

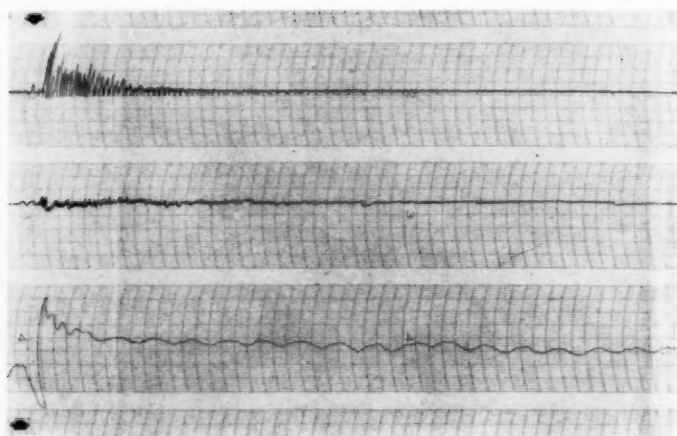


Fig. 7.—Clockwise rotation. The two upper graphs are photoelectric nystagmus recordings. The middle graph is calibrated as seen on the previous charts. The upper record is a derived nystagmogram according to Henriksson.¹⁵ Only the slow component of the nystagmus is recorded and the "eye speed" traced. The lower graph shows the lateropulsion. One large and two smaller inertia waves are seen after the chair stop. The total body deflection is 0.5 mm and the duration 25 sec. The waves along the record are caused by the respiratory motion of the thorax. Slight suppression of respiration follows the sudden stop of the chair.

for testing were healthy young males and females (medical and nursing students) with no history of ear disease, deafness or vertigo. Whereas, the great majority of the subjects experienced a variously long duration of sensation or illusion of motion after the rotating chair was stopped, none were aware of a lateral deviation of the body.

The records obtained show a series of characteristic phenomena (Figs. 5, 6, 7):

1. Lateropulsion occurs in every instance when postrotatory nystagmus is detectable.
2. A body deflection appears at the start of the rotation in the contralateral direction to the movement of the chair. During rotation, if the velocity is constantly maintained, no deviation is detected. After sudden cessation of turning the deflection follows the direction

of the rotation, i.e., the slow movement of the postrotatory nystagmus.

3. The shape of the deflection of the start and stop are different, due to the fact that the start of our chair is never as abrupt as is the stop. The total deviation in angles, however, is similar.

4. The actual deflection of the body is a very small motion. Applying a 45° per second velocity for 60 seconds, followed by a sudden stop, the deflection of the body in the majority of our normal, young individuals was less than 1 mm. It was as little in many instances as 0.2 or 0.3 mm, and the recorded maximum did not exceed 2.5 mm.

5. The average less than 1 mm postrotatory body deflection recovers surprisingly slowly. Considerable individual differences seem to exist. The recording pen drifts slowly and gradually back to the baseline. The recovery time (total duration) of the deflection varied between 12 and 56 seconds, with an average of 27 seconds.

6. There is no direct relation between the magnitude (in angles) of the deflection and the recovery time. A larger deflection may have a similar recovery time to that of a smaller body deviation.

7. In relation to the postrotatory nystagmus, the duration of the lateropulsion supersedes the duration of the former. The time difference may be small but in some instances the ratio was 2:1 and in one instance 3:1.

8. At the stop of the chair, the "pulsigram" invariably exhibits a greater excursion of the body. The first large wavy motion is frequently followed by a much smaller one. Occasionally a third, even smaller wave follows. The entire phenomenon ends within one or two seconds. It is produced by the inertia of the body which at times is also apparent by the simple observation of the subject in the rotating chair. The vestibular lateropulsion itself can be traced and studied following this short episode.

9. A gradation in the postrotatory lateropulsion could be demonstrated by recordings made in the same test subject, by increasing rotatory speed. Four consecutive rotations were performed within one-half hour time intervals between two rotations. The rotating chair was set for $22\frac{1}{2}^\circ$, 45° , 90° and 180° per second speed, and the subject rotated for 60 seconds. After a sudden stop the following

average body deflection values were obtained in four normal test subjects: recovery time (duration of the postrotatory lateropulsion) at $22\frac{1}{2}^{\circ}$ /sec speed, 12 sec; 45° /sec speed, 19 sec; 90° /sec speed, 34 sec; 180° /sec speed, 61 sec. The angle of deviation, expressed in millimeters at the height of the xyphoid process of the thorax, was 0.25 mm, 0.3 mm, 0.41 mm and 0.55 mm, in successive order of the four mentioned speeds (Table I).

TABLE I
GRADATION OF POSTROTATORY LATEROPULSION
AT VARIOUS SPEEDS

ROTATORY SPEED	RECOVERY OF POSTROTA-	
	TORY LATE ⁿ OPULSION	ANGLE OF DEVIATION
$22\frac{1}{2}^{\circ}$ /sec.	12 sec.	0.25 mm.
45° /sec.	19 sec.	0.30 mm.
90° /sec.	34 sec.	0.41 mm.
180° /sec.	61 sec.	0.55 mm.

10. The belt was applied around the chest at the height of the xyphoid process. In some individuals, the respiratory motion of the thorax was inadvertently recorded with our sensitive strain gauges. Instead of the straight baseline, a regular wavy line appears on the recording paper. The regular shape of the waves presents no difficulty in reading and evaluating the pulsion graph. Identical points of the respiratory curve must be considered along the reaction. By this incidental finding we were able to observe the effect of vestibular stimulation upon respiration. It is quite evident that a regular respiration during the rotation is abruptly suppressed by the sudden stop of the chair. Not only the rate, but the depth of the breathing is reduced. In several instances when the perrotatory respiratory waves were observed only as shallow excursions, the postrotatory lateropulsion appeared as an entirely straight drifting line.

This constitutes a brief review of observations we have made on recorded postrotatory lateropulsion. In some instances the lateropulsion was also measured during and after thermic stimulation. There is clear evidence that following even the so-called "minimal thermic stimulation" (10 cc 68° water syringed in the ear in five seconds of time in the sitting position), a lateropulsion will occur.

A number of patients with various pathological conditions were also tested with simultaneous nystagmographic and pulsion recordings. We intend to report these findings in a later communication.

SUMMARY

To measure vestibular excitability or sensitivity the produced vestibular nystagmus is the yardstick of evaluation. The various central vestibular pathways and other vestibulomuscular reflexes could not be utilized for a satisfactory objective clinical test and measurement. During vestibular stimulation a change in the muscular tonus occurs. One manifestation of this vestibulomuscular reflex is a minute deflection of the body. A recording mechanism was devised to measure postrotatory lateropulsion. A graphic recording of the body deflection is carried out simultaneously with photo-electric recording of the reactive nystagmus. The records are made by utilizing strain gauges, a direct current amplification and another channel of the same oscillograph which draws the nystagmus record.

Fifty young healthy individuals were tested by applying various rotatory speeds. The pulsogram reveals various characteristic information. It appeared as a reliable expression of vestibular excitability and offers strong support to the evaluation of the simultaneously recorded nystagmus.

1853 WEST POLK ST.

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VI

EPITHELIAL NEOPLASMS OF THE LARYNX

A DEPARTMENTAL REVIEW

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The papillomas and squamous cell carcinomas of the larynx in the surgical material of the University of Michigan Medical Center, Department of Pathology, from July 1, 1946, to June 30, 1956, and the necropsy records from July 1, 1923, to June 30, 1956, were reviewed. During this period 73 papillomas and 340 squamous cell carcinomas were seen, and 49 necropsies for neoplasms of the larynx were performed.

PAPILLOMAS OF THE LARYNX

Papillomas of the larynx have been described as being of the viral, juvenile or the hyperkeratotic type. In the material reported the juvenile type was more common. The papillomas tended to be multiple, had a loosely organized but definite vascular and connective tissue stalk, and were covered by a hyperplastic squamous or transitional epithelium. The hyperkeratotic papillomas tended to be more sessile lesions and occurred only in the upper age group.

The age distribution by decades of the 73 patients with papillomas is shown in Table I. When the patient had papillomas removed on more than one occasion, the age given is that at which a papilloma was first removed.

Of the 73 patients with papillomas, all but four were of the juvenile type. The remaining four papillomas were hyperplastic sessile lesions. Three of the hyperkeratotic papillomas occurred in the 51 to 60 years age group and one in the seventh decade. There were 53 males and 20 females, sex ratio 2.65 to 1.

In three instances the papillomas showed progression from typical juvenile papillomas into carcinoma; all of these patients had had

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TABLE I
ANALYSIS BY DECADES OF THE AGE AT WHICH
PAPILLOMAS BEGAN IN THIS SERIES

YEARS	CASES
0 - 10	16
11 - 20	10
21 - 30	8
31 - 40	7
41 - 50	5
51 - 60	16
61 - 70	5
71 - 80	4
81 - 90	1
91 - 100	1
TOTAL	73

papillomas removed on several occasions. One was a female, 59 years old, who had first had symptoms and a papilloma removed eight months previously. In four subsequent biopsies during this eight month period, the epithelial changes continued to become more atypical and finally warranted a diagnosis of carcinoma in-situ.

The two males were 53 and 60 years old; one had had papillomas removed every few years for 22 years and finally developed infiltrating carcinoma in a papilloma. A cervical lymph node metastasis was found at the time of total laryngectomy and lymph node dissection. The other patient had infiltrating carcinoma in a papilloma after having benign papillomas removed on several occasions over an eight year period.

In the series of 73 papillomas, 15 had papillomas excised at this hospital on two or more occasions for a recurrence rate of 20 per cent. Twenty-one patients had had more than one papilloma removed on at least one occasion and ten had had multiple papillomas excised on two or more occasions.

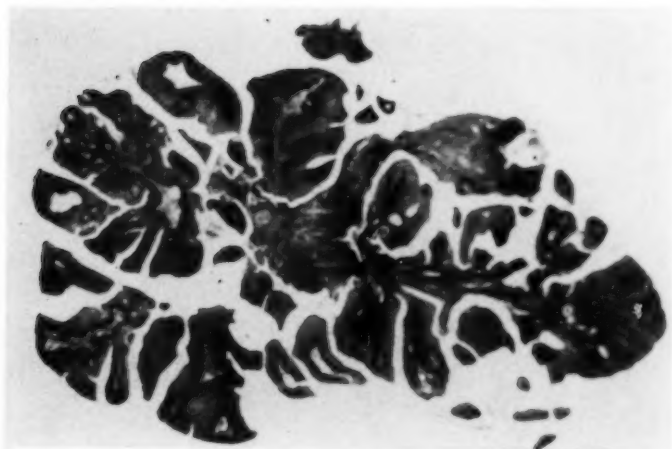


Fig. 1.—Juvenile papilloma of larynx. (Hematoxylin-eosin, X 20)

COMMENT

Papillomas of the larynx may occur at any age; however, the peak incidence in this series was in childhood and in the sixth decade. Similar age distribution patterns have been noted by other observers. In Rubin's¹ series of 26 cases, no papillomas were present in the 18 to 32 year old group and the highest incidence occurred in the 40 to 60 year old group.¹ Fifty-four of the 109 cases of papillomas reported by Holinger et al^{2,3} occurred before the 16th year of age.

All of the series reviewed showed a greater incidence of papillomas in males.¹⁻⁴ The ratio of males to females was 12:1 in Rubin's series, 1.18:1 in Holinger's report, and 1.8:1 in del Villar's series.⁴

The fact that carcinoma may arise in papillomas makes the continued clinical surveillance and histologic examination of such lesions imperative. The three patients in this series represent an incidence rate of 2.5 per cent which is a high rate of malignant change in lesions that are generally considered to be benign. Other authors noted a high rate of malignant transformation in these lesions.^{1,5-9} Rubin reported two of 26 patients which showed malignant change. New and Erich⁵ found malignant change in three of 194 papillomas, and Jackson and Jackson⁷ found carcinoma in six of a series of 205 papil-

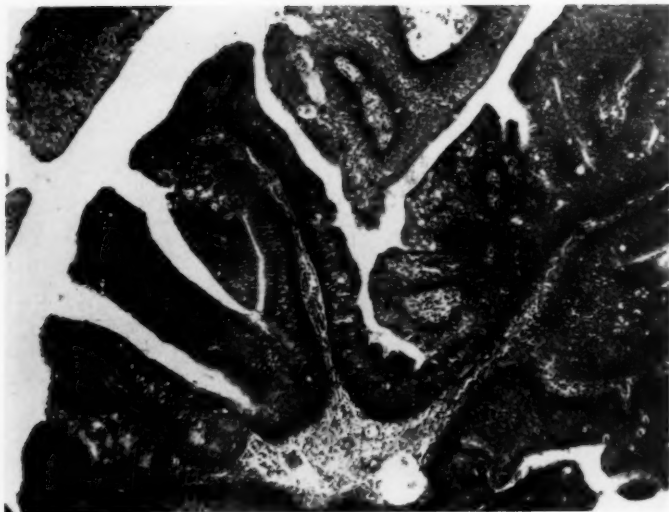


Fig. 2.—Juvenile papilloma of larynx. Epithelium covering delicate fibrous tissue stalks. Respiratory type, columnar, transitional, and squamous epithelium. (Hematoxylin-eosin, X 93)



Fig. 3.—Squamous and transitional epithelial covering of a juvenile papilloma of larynx. (Hematoxylin-eosin, X 261)

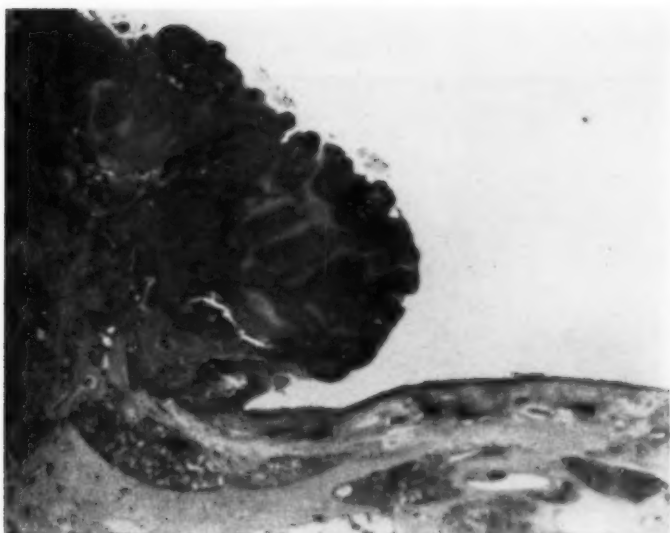


Fig. 4.—Recurrent juvenile papillomas with carcinomatous change, broad anastomosing cords of squamous carcinoma cells are present in the lesion. (Hematoxylin-eosin, X 23)

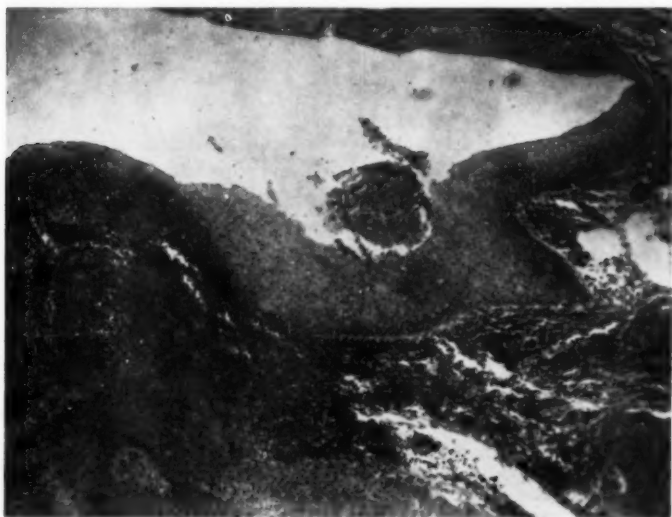


Fig. 5.—Carcinoma in Figure 4 showing the zone of transition from squamous epithelium of juvenile papilloma to invasive carcinoma. (Hematoxylin-eosin, X 92)

TABLE II
GRADES OF THE CARCINOMAS OF THE
LARYNX IN THE SURGICAL SERIES

Carcinoma in situ	7
5 males - 2 females (average age, 57 years)	
Grade I	6
5 males - 1 female (average age, 53 years)	
Grade II	177
161 males - 16 females (average age, 60 years)	
Grade II-III	6
All males (average age, 54 years)	
Grade III	136
126 males - 10 females (average age, 63 years)	
Grade III-IV	2
Males (ages, 45 and 57 years)	
Grade IV	6
4 males - 3 females (average age, 60 years)	

lomas in adults. Walsh⁶ reported two carcinomas arising in papillomas in children. One was a 12 year old boy who had had papillomas in the larynx for eight years. The second was a girl who had papillomas at three years of age and developed carcinoma in a papilloma at ten years of age.⁶

Putney⁸ reported that nine carcinomas developed in a series of 501 papillomas. The interval between the benign and malignant lesions varied from one to 23 years. Stout⁹ reported nine carcinomas in situ arising in recurrent papillomas in 312 laryngeal carcinomas.

Ten patients in this series had multiple papillomas excised on more than one occasion, and eight of the patients were less than 25 years of age. In the two patients in whom the lesions occurred in later life, carcinoma eventually developed. The third patient with carcinoma arising in a papilloma had solitary papillomas excised on several occasions.

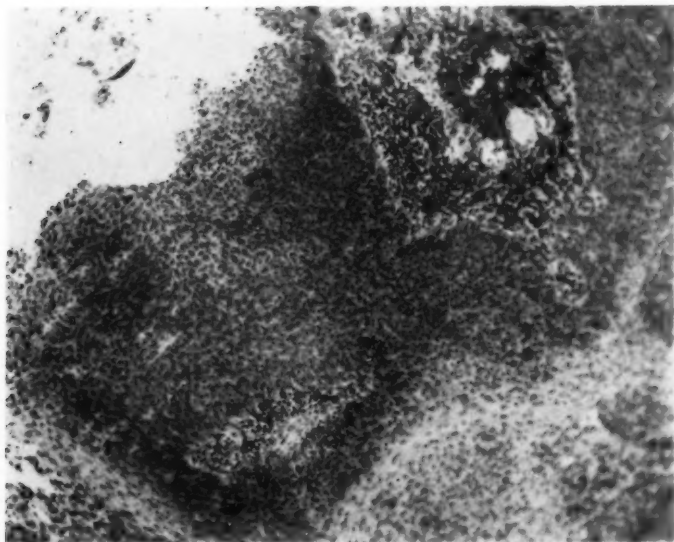


Fig. 6.—Carcinoma seen in Figure 5. Note the loss of stratification and polarity of the epithelial cells. A few mitotic figures are present. Lymphocytic infiltration is present in the stroma, as in the benign lesion (Fig. 3). All of the carcinomas arising in papillomas showed this type of noncornifying squamous epithelium. (Hematoxylin-eosin, X 225)

Papillomas occurring in the 50 to 60 year age group and multiple papillomas occurring in adults would appear to warrant more frequent clinical surveillance than is necessary for histologically benign lesions.

CARCINOMA OF THE LARYNX

During a ten year period 340 carcinomas of the larynx were examined in surgical material. The carcinomas were graded as carcinoma in situ, and grades of I to IV depending on the differentiation, were assigned to the infiltrating carcinomas. To warrant a diagnosis of carcinoma in situ, the cellular atypicalities had to be those of carcinoma; however, the lesion was confined to the mucosa. When the basement membrane was infiltrated the lesion was classified as infiltrating carcinoma.

Grade I carcinomas were early, well differentiated, but infiltrating lesions. Grade II carcinomas were well differentiated and showed



Fig. 7.—Carcinoma in situ of larynx. The epithelium is hyperkeratotic and dyskeratotic. Despite the nuclear atypicalities the basement membrane is intact. (Hematoxylin-eosin, X 85)

keratine production. Grade III carcinomas had noncornifying cells. In Grade IV carcinomas the cells were atypical and showed little evidence of having arisen from squamous epithelium. In several instances these carcinomas assumed a spindle-celled pattern.

In a few instances no single grade was assigned to the carcinoma since different fields presented different histologic patterns. In the tabulation of results, these were reported, Grade II to III or Grade III to IV, depending on the degree of differentiation.

Table II indicates the histologic grade of the neoplasms and average age of 340 patients with laryngeal carcinoma.

COMMENT

In the surgical material 17 lesions were reported as carcinoma in situ; however, on examination of additional material, 10 patients showed foci of infiltrative carcinoma, leaving only seven carcinomas in situ. Other authors have noted that infiltrative carcinoma existed with carcinoma in situ.^{8,9} Stout⁹ reported 29 cases of intraepithelial

TABLE III
GRADES OF CARCINOMAS IN THE
NECROPSY SERIES

Grade I	8
5 males - 3 females (average age, 54 years)	
Grade II	37
36 males - 1 female (average age, 62 years)	
Grade III	2
Both males (average age, 61 years)	

carcinoma, and 19 more in which invasive carcinoma was present in addition to the carcinoma in situ. The average age of patients with carcinoma in situ of the larynx in our series was 57 years. This age was in agreement with other series reported in the literature.⁹⁻¹¹ In carcinoma of the cervix, the average age for in situ lesions is about ten years earlier than infiltrative carcinoma.⁹ In all series reviewed, more males than females were reported with carcinoma in situ of the larynx.⁹⁻¹² Stout's series was the largest reviewed in the literature, a ratio of 12 males to 1 female.

The poorly differentiated carcinomas of larynx occurred in only a slightly older age group than did the well-differentiated carcinomas. The three patients with carcinoma arising in papillomas were at the age of peak incidence of carcinoma. The average age in this series agreed with other reports in the literature,¹³⁻¹⁷ a ratio of 10.5 males to 1 female. Baltzell¹⁴ had a male to female ratio of 13 to 1; Wynder et al¹⁸ 10 to 1; Kirchner and Malkin^{15,16} 12 to 1.

EPITHELIAL NEOPLASMS IN THE NECROPSY SERIES

The University of Michigan Medical Center necropsy material was reviewed from 1923 to 1956. Forty-seven carcinomas, one papilloma, and one papillary cystadenoma were found. During this period 14,000 necropsies were performed, and carcinoma of the larynx constituted 0.3 per cent of all necropsies. Malignant neoplasms were present in 29 per cent of the necropsies. Carcinoma of the larynx constituted 1 per cent of the necropsies with malignant neoplasms. The sex ratio was 10.7 males to 1 female, comparable to the ratio in the surgical material.



Fig. 8.—Cornifying squamous cell carcinoma, infiltration adjacent to laryngeal cartilage. (Hematoxylin-eosin, X 110)

The laryngeal carcinomas were graded by the same method used in the surgical material. Table III shows the frequency of each grade and average age of the patients at time of death. None of the Grade I lesions showed infiltration into extralaryngeal tissues or metastases. Of the 37 Grade II lesions, 24 showed metastases and infiltration into extralaryngeal tissues, one showed infiltration into extralaryngeal tissues alone, and 12 showed no extralaryngeal involvement. Both of the Grade III carcinomas showed metastases and infiltrations into extralaryngeal tissues.

Table IV shows the frequency and site of infiltration and metastases in the necropsy series; Table V shows the cause of death from carcinoma of the larynx.

TABLE IV
METASTASES AND INFILTRATIONS OF
CARCINOMA OF THE LARYNX
IN THE NECROPSY SERIES

INFILTRATIONS	
Local extension to voluntary muscle	14
Thyroid	8
Trachea	6
Esophagus	7
Tongue	1
Tonsils	1
Parotid gland	1
METASTASES	
Cervical lymph nodes	13
Tracheal-bronchial lymph nodes	4
Lungs	3
Pleura	2
Liver	1
Adrenal gland	1
Bone	1
Thymus gland	1
Submaxillary lymph nodes	1

TABLE V
CAUSE OF DEATH IN 47 PATIENTS
WITH CARCINOMA OF THE LARYNX

Pneumonia	25
Suffocation	5
Cachexia	4
Lobar pneumonia	1
Tuberculosis	1
Septicemia (wound infection)	1
Coronary arterial occlusion	2
Uremia	1
Peritonitis (perforated appendix)	1
Gastrointestinal hemorrhage	1
Rheumatic heart disease	1
Arteriosclerosis	2
Pulmonary infarct	1
Myocardial infarct	1

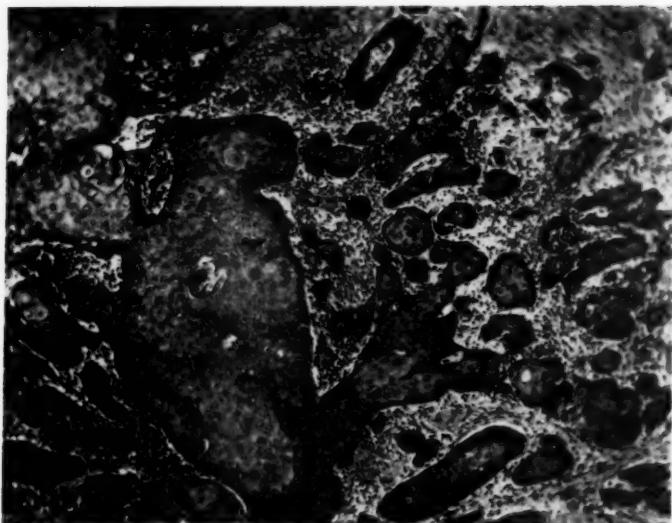


Fig. 9.—Squamous cell carcinoma from the larynx showing slight cornification. (Hematoxylin-eosin, X 110)

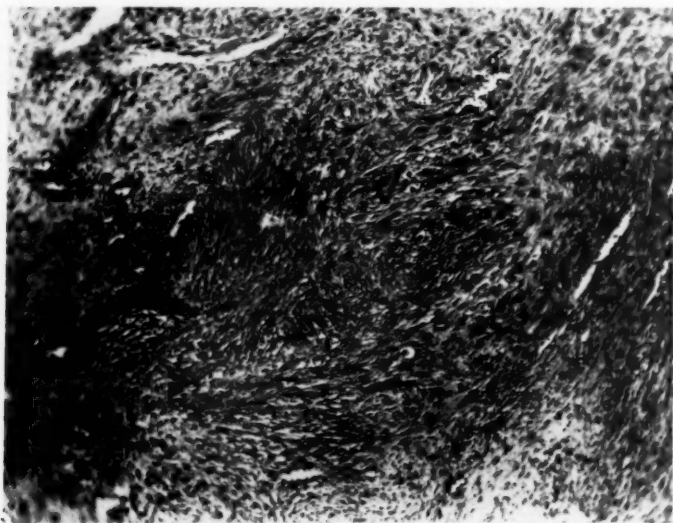


Fig. 10.—Carcinoma of the larynx showing a spindle celled pattern. (Hematoxylin-eosin, X 165)

COMMENT

The grade of the carcinoma, average age of the patients at time of death, and sex ratio corresponded with the data from the surgical material.

The distribution of sites of infiltration and metastases emphasizes the fact that squamous cell carcinoma usually remains well-localized to the neck structures even at time of death. Sawyer et al¹⁹ reviewed the literature from 1878 to 1947 and reported 14 instances in which multiple blood borne metastases were present, but only eight of these cases were examined histologically.¹⁹ One of the patients in the series reported here exhibited blood borne metastases with involvement of the left adrenal gland, lungs, and pleura.

The thyroid gland was invaded in eight of our 47 cases. Ogura²⁰ found invasion of the thyroid in six of 59 patients. Thirteen of our 47 carcinomas showed cervical lymph node metastases. In one series reported in the literature 76 patients were found to have lymph node metastases in 441 specimens from radical neck dissections.²¹

Pneumonia was the most frequent cause of death in this series; however, suffocation was the most frequent cause of death directly attributable to the neoplasm. In several cases of cachexia, extension of the laryngeal carcinoma into the esophagus was present. In one patient septicemia was due to a suppurative wound infection at the site of laryngectomy.

Eight of the 47 patients with laryngeal carcinomas had double primary malignant neoplasms. The second primaries were: squamous cell carcinoma of the bronchus, 2; leukemia, 2; adenocarcinoma of the prostate, 1; and one each, squamous cell carcinoma of the abdominal wall, cervix, and esophagus. The incidence of double primaries in this series approximates that of Wynder and Bross²² who found 21 double primaries in a series of 209 cases of carcinoma of the larynx. Eighteen of the double primaries in their series involved the lung, esophagus, or oral cavity. They felt that this indicated a common etiologic factor in these neoplasms.²²

The one papillary cystadenoma in this necropsy series was an incidental finding. According to Ranger and Thackray²³ less than 20 cases have been reported in the literature.

SUMMARY

1. A review of the epithelial neoplasms of the larynx in the University of Michigan Medical Center surgical and necropsy material is presented.

2. Juvenile papillomas occurred about as frequently in the upper age group as in children.

3. Carcinoma may develop in the juvenile papilloma, especially in the 50 to 60 year age group.

4. Carcinoma in situ occurs at approximately the same age as invasive carcinoma and frequently coexists with invasive carcinoma.

5. Hematogenous metastases from carcinoma of the larynx is unusual.

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VII

CEREBROSPINAL OTORRHEA

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(by invitation)

CHICAGO

Escape of cerebrospinal fluid from the ear is indicative of pathologic communication between the subarachnoid space and the temporal bone. Leakage is usually a result of temporal bone fractures, and occasionally follows surgical trauma or infection. However, in the absence of trauma or infection, and in the light of present knowledge of temporal bone embryology, the rare possibility of congenital dehiscences responsible for "liquor tympanum" must be considered.

The purpose of this presentation is to discuss the conditions responsible for cerebrospinal otorrhea, the mechanism involved, and the complications which may ensue, and to develop further the concept of a congenital pathogenesis.

Fractures of the temporal bone accompanied by dural tears, are the most common cause of cerebrospinal otorrhea. Distinction must be made between instances in which there is an intact tympanic membrane and those in which the membrane is ruptured. The latter, occurring in the presence of an existing otitis media, is likely to spread infection into the subarachnoid space, with resultant meningitis.

The most common type of fracture encountered is the longitudinal, or middle fossa fracture, in which the course of the break is parallel to the long axis of the petrous pyramid. Such a fracture usually passes in front of, or at the level of the external auditory canal, and terminates at the anterior foramen lacerum in the region of the Gasserian ganglion. The middle ear is almost always injured, usually with a lacerated tympanic membrane. The incus may become dislocated, and on occasions, facial paralysis may result.

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Interruption of continuity in the ossicular chain results in moderate to severe conductive hearing loss, and occasionally in vertigo. The bony labyrinth is usually spared, unless there is also a transverse fracture. Fissures are frequently found between the windows; the footplate of the stapes is often luxated, and the promontory shattered.

Transverse temporal bone fractures are usually found at right angles to and through the pyramid, with consequent damage to the membranous labyrinth. The tympanic membrane is rarely ruptured, but the severance of nerves in the internal auditory meatus is not infrequent. Injury to the cochlear and vestibular components is the rule, with resultant complete loss of function.

Important to bear in mind, are the microscopic fractures which are not radiographically visible but may have serious implications in the presence of infection, either primary or secondary. Danger of meningitis, early or late, is always to be considered whenever open communication exists between the subarachnoid space and the middle ear, and injuries without obvious intracranial impairment must not be regarded with complacency.

Persistent cerebrospinal leakage from the ear will tend to lower intracranial tension and facilitate the formation of intracranial blood clots. Cessation of the drainage may result in increased intracranial pressure and subsequent coma. The brain, although displaced by hemorrhage or hematoma, may not be compressed, so that its function remains intact as long as the cerebrospinal fluid escapes.

Pneumocephalus and cerebrospinal fluid fistulas are manifestations of the same underlying pathologic state. The presence of air in the intracranial cavity as seen in the roentgenogram is conclusive proof of a dural tear associated with the temporal bone fracture. When undoubted evidence exists, dural repair by the use of fascia lata grafts can be accomplished. This type of repair applies to dural tears associated with fractures of the middle ear and mastoid as well as those of the anterior fossa.

The history of injury with associated cerebrospinal otorrhea and presence or absence of pneumocephalus is incontrovertible evidence of dural rupture. Cerebrospinal fluid from the ear can be identified by analysis of its sugar content (sugar reducing test). However, this analysis is unreliable when, in recent injuries, discharging cerebrospinal fluid is combined with blood or serum. An adjunct to diagnosis, as was carried out in the case being presented, is the instillation of a dye

into the spinal canal and observation of its emission from the ear. Through this technique one may be able to locate the site of the fistula.

The relationship between meningitis and trauma, particularly after an interval of many years, may be established only after searching inquiry. Careful roentgenologic studies may provide significant information, but the exact site of the fracture and dural tear may not be known until exploration is undertaken.

REPORT OF A CASE

A fifteen-month old white female was first observed in December, 1955, having been referred to the Otolaryngologic Clinic of the Illinois Research and Educational Hospitals of the University of Illinois because of watery discharge from the right ear.

According to the history, the drainage became evident in July, 1955, unrelated to trauma, either during delivery or later, or to infection. The discharge which was clear, continued unabated. Although no fever was present, the patient had previously been treated by a local physician with antibiotics, and the ear packed with cotton. The watery discharge ceased within a few days. However, in September, 1955, the drainage recurred, and Terramycin ear drops were employed, and once again the discharge ceased, only to return one month later.

The child was admitted to a private hospital for observation. X-ray and spinal fluid studies were within normal limits. Pneumoencephalogram and surgery were recommended, but were refused by the family. The patient was sent home where she developed a fever of 104° F. without other signs. Antibiotics were given and within 24 hours the temperature was normal.

Three weeks prior to admission to the R. & E. Hospitals, the patient became hyperirritable and began losing weight, although to the time of admission she had enjoyed otherwise good health.

She was admitted because of the watery discharge from the right ear. Examination was essentially normal except for the presence of a reddish mass in the external auditory canal, which appeared to be arising from the superior wall of the middle ear. A clinical diagnosis of meningocele was entertained. X-rays of the mastoid revealed a questionable dehiscence of the tegmen tympani on the right. Spinal fluid examination and EEG were normal. The sugar reduction test

(Benedict) of the fluid was positive. Culture studies revealed *Pseudomonas*.

On January 16, 1956, an endaural approach was carried out. The mastoid and middle ear were found to be within normal limits. The ossicles appeared normal, and there was no evidence of a meningocele or dehiscence in the tegmen. Indigo carmine, 0.8%, was introduced via spinal tap, and within a few minutes cerebrospinal fluid (blue) was seen flowing from around the oval window. Temporal fascia was used to cover the dehiscence and a pressure packing was applied. The packing was removed ten days later and cerebrospinal fluid was observed coming from the previously-described site. Skin grafting was performed, but to no avail.

A right posterior temporal craniotomy was carried out on June 18, 1956. Exposure of the area revealed a vein-like structure in the area of the upper posterior surface of the petrous bone which appeared to invade the bone. This was coagulated with diathermy and packed with gelfoam. One week later the cerebrospinal otorrhea recurred.

On August 1, 1956, a posterior fossa exploration was again performed and since there was no evidence as to where the defect might be, the internal auditory meatus was packed with gelfoam. Since this, there has been complete cessation of otorrhea, and the child is well. There is complete absence of vestibular and cochlear junction in the right ear.

COMMENT

Since the history elicited did not reveal trauma or infection that might be held responsible for the cerebrospinal otorrhea, and in the absence of obvious fracture or fistula at the time of the intracranial approach, the possibility of a congenital defect is suggested. It is quite possible that there may have been a trauma of which the family is unaware. Also, a dehiscence may have been present, which became obvious following an infection.

However, in view of the numerous surgical procedures performed without evidence of a defect, and the cessation of the drainage following internal auditory packing, a congenital etiology is to be considered.

The anatomic and embryologic relationship to cerebrospinal otorrhea requires elucidation to better understand the pathogenesis

of congenital origin. Both the subarachnoid and subdural space extend into the internal ear and indirectly into the brain:

1) *Cochlear Aqueduct.* In both children and adults the intermeningeal spaces extend deep into the cochlear aqueduct. Extension of both spaces within the aqueduct varies from case to case. Frequently the connective tissue of the subarachnoid space extends up to the scala tympani of the vestibular portion of the cochlea where it blends with the periosteum of the scala. Although from the anatomic point of view the cochlear aqueduct furnishes a free communication between the perilymph of the internal ear and the cerebrospinal fluid of the subarachnoid space, in adults the channel is apparently too narrow to encourage a liberal mingling of the two fluids.

2) *Internal Auditory Meatus.* The intermeningeal spaces extend into the internal auditory meatus. The auditory nerve within the meatus is surrounded by an arachnoid sheath which, at the fundus of the meatus, blends with the dural periosteum of the channel. Where the nerve enters the cavity of the skull, the arachnoid blends with the external layers of the arachnoid of the cisterna pontis and the nerve runs through the cisterna pontis toward the medulla oblongata. Another anatomic point of interest is the proximity of the cisterna pontis lateralis to the petrous bone, extending from the apex toward the posterior surface of the petrous bone where the internal auditory meatus and cochlear aqueduct drain into the cisterna.

3) *Periotic Space Around Otic Duct.* The otic (endolymphatic) duct extends from the point of union of the saccular and utricular ducts, through the otic capsule, to a termination in the otic sac. The otic duct and sac are not surrounded by the periotic labyrinth except proximally, where a short, funnel-shaped diverticulum of the vestibule envelops the otic duct as the latter enters the vestibular aqueduct of the otic capsule. This periotic infundibulum surrounds the otic duct for only a short distance into the intracapsular aqueduct; its derivation represents a spread of the process of vacuolization in the vestibule. The remainder of the connective tissue by which the otic duct and sac are enveloped, possesses no periotic relationships; it assumes definitely meningeal character the external (cranial) aperture of the aqueduct.

SUMMARY

Cerebrospinal otorrhea is discussed both from a traumatic and congenital point of view. The pathogenesis of a congenital etiology is

elaborated upon, based on anatomical and embryologic fundamentals. Diagnosis and management are considered, as well as the complications associated.

DISCUSSION

DR. PAUL R. ROSENBLITH (by invitation): We, in neurosurgery, are particularly grateful for this type of lesion because it occasionally results in pneumocephaly, and it was such a case that suggested to Dr. Dandy at the Johns Hopkins Hospital in 1918 that air could be introduced into the chambers of the brain for diagnostic purposes, and from this resulted the techniques of ventriculography and pneumoencephalography.

The pathology of basilar skull fracture with otorrhea was first described in a Paris clinic in 1839 when it was perceived that there was a watery discharge from the ear in certain cases of severe head injury. Autopsies disclosed fracture of the petrous bone and rupture of the tympanic membrane. Having correctly noted the pathological fracture, however, the conclusion was that the watery fluid represented serum from extravasated blood, it not having been recognized as cerebrospinal fluid.

Dr. Ferrer described the present case as a small petrous-acoustic fistula. Many of these fistulas heal spontaneously. Spontaneous healing is encountered particularly in otorrhea following fracture of the petrous bone due to the long course of fluid and relatively greater thickness of the bone and soft tissue as compared to drainage through the nose. Such post-traumatic discharge rarely requires operative intervention. Occasionally otorrhea may be manifested as a rhinorrhea when the tympanic membrane is not perforated and the fluid drains through the nose, or through the pharynx. Such injury rarely requires operative intervention.

In diagnosing the presence of cerebrospinal fluid, the simplest test is, of course, Benedict's sugar-reducing test. In the absence of this, there is the so-called "Handkerchief-stiffening test." Spinal fluid will not stiffen the material, while other secretions will. Sometimes it is necessary to inject a dye such as indigo carmine or methylene blue into the subarachnoid space. A cautionary word should be added, because there have been several cases reported of severe reaction to these dyes, and the rare occurrence of transverse myelitis after the injection of methylene blue. Of course, there is no point in using indigo carmine when there has been recent bleeding.

Any persistent cerebrospinal fistula must be repaired, for there is always the threat of meningitis.

VIII

EXPERIMENTAL OBSERVATIONS ON POSTURAL NYSTAGMUS

II. LESIONS OF THE NODULUS

CÉSAR FERNANDEZ, M.D.

RENÉ ALZATE, M.D.

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CHICAGO, ILL.

The interrelations between vestibular system and flocculonodular lobe have been demonstrated by anatomical and electrophysiological methods. The publications of Jansen and Brodal¹ and Dow and Moruzzi² covered extensively the literature in this area.

The anatomical studies of the cerebellum revealed that the flocculonodular lobe is phylogenetically the oldest division. It develops from the vestibular nuclei and its anatomical configurations have been maintained throughout the phylogenetic scale, although its size varies among species. Its afferent and efferent connections are mainly with the vestibular system. Jansen and Brodal¹ emphasized that the fiber connections of the nodulus are not equivalent with those of the flocculus, which may account for difference in results following ablation of one segment or the other.

The ablation experiments which included nodulus alone or nodulus and adjacent segments of the paleocerebellum^{3,7} revealed a syndrome characterized by disturbance of both equilibrium and eye movements. The pattern of disequilibrium consists in swaying of the body from side to side when either standing or moving. The gait is unsteady and broad-based. Falling frequently occurs. Unlike the deficit produced by lesions in other regions of the cerebellum, there is no disturbance in posture nor in range, rate, force and direction of volitional movements.

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There is little or no disagreement among investigators with respect to these signs of disequilibrium. This is not the case, however, in so far as patterns of eye movements are concerned. For example, after lesions in the nodulus, spontaneous nystagmus has been reported in the guinea pig,⁶ postural nystagmus in the cat,⁵⁻⁸ but neither spontaneous nor postural nystagmus have been observed in the monkey³ and dog.⁹ In man, lesions in the posterior vermis can produce postural nystagmus;¹⁰⁻¹⁴ a finding which is of considerable diagnostic importance.

The discrepancy in the literature regarding the occurrence and form of nystagmus after ablation of the nodulus prompted this investigation. In addition, the effects of deafferentation of vestibular centers upon disturbances of both equilibrium and eye movements were also studied.

METHOD

Twenty-four healthy adult cats served as subjects. The observation consisted in exploring the responses before and after removal of the nodulus on the following tests.

Standing and Motor Performance. This included attitude, gait and performance of fine movements such as, jumping in or out of the animal cage, grasping a small object and jumping from a chair. Special care was taken to differentiate signs of disequilibrium from signs of cerebellar ataxia.

Spontaneous Nystagmus. For observing spontaneous nystagmus the cat was sustained in normal position with the head gently fixed between the hands of the observer. Suitable movements of objects in the animals' visual field permitted examination of the lateral gaze.

Postural Nystagmus. The animals were tested for postural nystagmus according to the procedure which is followed in clinics, that is, the eye movements were observed in supine, lateral right, lateral left and hanging head positions. In some animals the abnormalities were recorded by means of nystagmography.

Righting Reflexes. The animal was held upside down at about two feet above a cushion and then dropped in a free fall. The test was done with both eyes open and eyes closed.

Rotatory Test. The animal was immobilized in a cast specially made to prevent movements of head and trunk. The cast was fixed

in a rotating device. In some animals the number of jerks and duration of the postrotatory nystagmus were determined directly by the observer, while in others the postrotatory reactions were recorded by nystagmography. The rotating table was accelerated at 2.0 degrees per second squared for 30 seconds, followed by a rotation at constant angular velocity for 30 additional seconds. Deceleration was done in one and one-half seconds. The animal was blindfolded and rotated first to the right and after an interval of five minutes was rotated to the left. Because the immobilization of head was not complete, the nystagmographic records were often contaminated with head movements.

Operative Procedure. The operation consisted in exposing the posterior cerebellar vermis through a suboccipital craniotomy approach. The uvula was gently elevated and the IV ventricle opened. The choroid plexus was partially sucked out occasionally producing a transitory hemorrhage. Further elevation of the uvula permitted exposure of the nodulus. This was removed by suction with a fine pipette until the surface of the lingula appeared. In most instances the ablation was intended to be complete. In all animals care was taken to avoid injury to the rhomboid fossa, cerebellar peduncles and other cerebellar segments. Elevation of the uvula produced, in some cases, traumatic petechial hemorrhages in its cortex. The IV ventricle was cleaned of blood as much as the circumstances permitted. No suture of the dura was intended and the bony defect was closed with gelfoam.

The animals were maintained under observation for periods of seven to 70 days. They were sacrificed by intravital fixation with 80 per cent alcohol. The cerebellum together with the brain stem were treated for Nissl's stain. The serial sections included the whole cerebellum and brain stem. Every twentieth section was stained and studied under regular light microscopy.

The histologic examination revealed that the uvula was always encroached upon; consequently, in describing the effects of lesions in the nodulus, it should be understood that this operation invariably produced some damage to the uvula.

RESULTS

I. ABLATION OF THE NODULUS

This operation was done in 12 cats. The results demonstrated a clear cut syndrome characterized by disturbances of both equilibrium

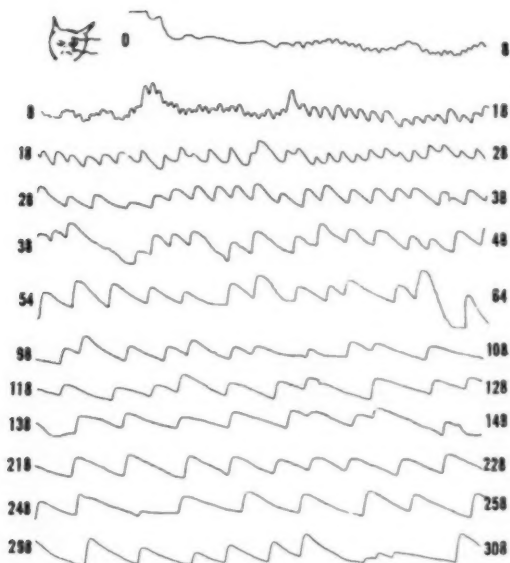


Fig. 1.—Cat A-596. Nystagmograph taken in hanging head position three days after ablation of the nodulus. The figure is composed of twelve samples taken during the first five minutes, and shows a persistent type of postural nystagmus. Numerals indicate time in seconds since change in body position. In this and the following nystagmographs, unless otherwise specified, the electrodes were placed in a vertical plane and the direction of the nystagmus was always toward lower eyelids. The graphs represent a continuous recording, reading from left to right; numerals indicate time in seconds and the instant the animal was rotated from a standing position to a new position is signified by a zero mark.

and eye movements. For convenience and a better understanding the over-all result of each test will be described separately.

Standing and Motor Performances. Twenty-four hours after the operation the animal could sit up supported by its forelegs. The head was held in erect position. The body would sway and not infrequently the animal would fall. No static tremor was ever observed. Locomotion was resumed in about 36 hours but showed profound equilibratory disturbances. During forward progression the gait was broad-based with the animal deviating markedly from side to side. This resulted in falling on a number of instances. Some cats consistently walked with limbs extended. The appraisal of motor perform-

ance by direct observation and motion pictures was that no abnormalities in range, rate, force and direction of movements were present; in other words, no signs of cerebellar ataxia could be detected. The signs of disequilibrium subsided gradually within 20 to 25 days; thereafter, standing and motor performance appeared normal except fine movements. Spontaneous shaking of the head and jumping from the floor into home cage remained disturbed for a much longer time.

Spontaneous Nystagmus. This sign was not seen in our series of animals after complete recovery from anesthesia. During recovery spontaneous nystagmus occasionally occurred, but this was attributed to the anesthesia.

Postural Nystagmus. In addition to disequilibrium the most striking finding was the appearance of postural nystagmus. It was consistently observed in supine and hanging head positions and was frequently seen in lateral right or lateral left positions or both. It was always direction-fixed, i.e., it occurred in one direction regardless of the position. In every case the direction was vertical toward the lower eyelids; however, in a few instances, the nystagmus exhibited a rotatory component. Regarding the other characteristics of nystagmus (latency, amplitude, frequency and duration) several different patterns were observed. In some animals the onset of postural nystagmus was delayed and the course was in rapid crescendo and decay, but it remained present as long as the position was maintained. A representative case is illustrated in Figure 1. This persistent type of postural nystagmus may become paroxysmal in time. In most animals the postural nystagmus was typically paroxysmal. That is, the latency of nystagmus was sometimes as long as 50 seconds; the onset was preceded by struggling, swallowing and vocalization; the frequency rapidly increased to a maximum, then declined and finally disappeared within 30 to 180 seconds. Unfortunately in most cases, the nystagmography was contaminated by artifacts introduced by struggling and blinking. A fair recording of this paroxysmal type of postural nystagmus is presented in Figure 2. The latency was 38 seconds, the course was in rapid crescendo, then decayed and disappeared in 100 seconds. Figure 3 illustrates another pattern of postural nystagmus in supine position two days after ablation of the nodulus. In this case there was no measurable latency, nystagmic outbursts appeared from time to time and the duration was about 120 seconds.

The question whether the postural nystagmus would habituate itself with daily sessions of repetitive postural tests was investigated in many cats, but the results were variable. Habituation occurred

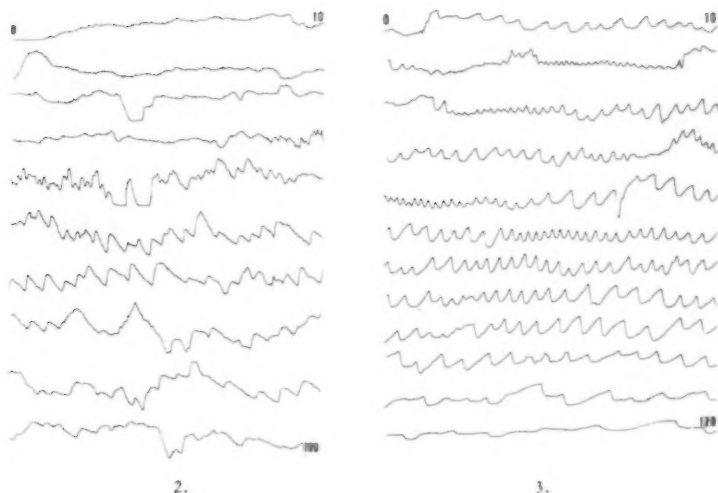


Fig. 2.—Cat A-660. Nystagmograph taken in supine position three days after ablation of the nodulus, illustrating the paroxysmal type of postural nystagmus. See text for a description of its characteristics.

Fig. 3.—Cat A-674. Nystagmograph taken in supine position two days after ablation of the nodulus. The absence of nystagmus at the beginning of the record is due to time required for turning the animal from normal standing to supine position. Once in this position, the onset of the nystagmus was immediate and lasted for 120 seconds. Notice the recurrence of nystagmic outbursts. In hanging head position, on the other hand, this animal has a postural nystagmus with a definite latency.

in some cases but not in others. Perhaps small differences in the size of the lesion may have been responsible.

Compensation for disturbances of eye movements usually required about ten days, but in two animals postural nystagmus persisted for twenty days.

Righting Reflexes. These reflexes were not modified by the ablation of the nodulus.

Rotatory Test. The removal of the nodulus did not modify significantly the characteristics of postrotatory nystagmus (Fig. 4). The records show that at 24 and 48 hours after ablation the duration was not altered while amplitude, in the first postoperative day was larger.

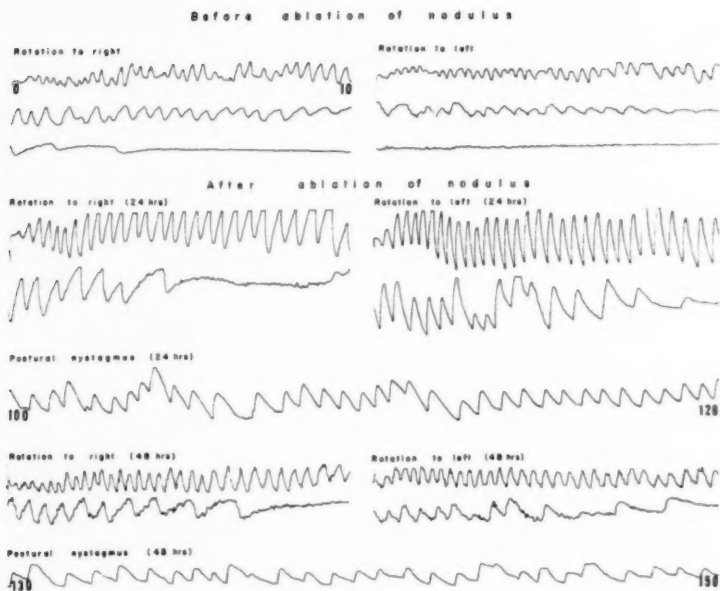


Fig. 4.—Cat A-728. Effect of ablation of nodulus on postrotatory nystagmus. The records demonstrate that the duration of postrotatory nystagmus was not changed by the operation while intensity and frequency of postural nystagmus was at the peak. The postural nystagmus was a persistent direction-fixed type. The large amplitude of postrotatory nystagmus on the next postoperative day may be due to mechanical arrangement of electrodes. Small variations in the placement of electrodes, position of the head and other causes in the cat may produce changes in the amplitude of the nystagmograph.

This may be due to the differences in mechanical arrangement of the electrodes. In a few animals, however, the rhythm of postrotatory nystagmus was abnormal for one or two days. Figure 4 also shows that at 24 and 48 hours, the postural nystagmus was present. The sample was taken from records in supine position. The postural nystagmus was of a persistent direction-fixed type.

Histologic Examination. The serial sections including cerebellum and brain stem revealed that the nodulus was never entirely removed (Figs. 3 and 6). All specimens exhibited partial ablation or cellular degeneration or both in some areas of the uvula. This cellular degeneration seems to be the result of surgical petechial hemorrhages. No

involvement of the rhomboid fossa, cerebellar nuclei and cerebellar peduncles was ever found.

The correlation between extension of the lesion and severity of responses seems to indicate that when the lesion was small (Fig. 7) the disturbances in equilibrium and eye movements were mild while severe disturbances were associated with extensive lesions.

Comment. The data on this series of animals with ablation of the nodulus confirmed previous observations regarding disturbances of equilibrium. In addition, they disclosed abnormal patterns of eye movements that may have been overlooked by some of the earlier investigators. The abnormality in eye movements was postural nystagmus without spontaneous nystagmus.

Although disturbances in equilibrium with abnormal eye movements take place after lesions in the nodulus, it can be argued that these effects may be brought about by a transitory disturbance of the adjacent cerebellar or medullary structures, or both.^{5,15-17} Our data support the notion that it is the lesion in the nodulus that produces disequilibrium and postural nystagmus. More specifically control experiments were carried out in which the IV ventricle was opened to simulate the surgical approach for removal of the nodulus. The choroid plexus was partially sucked out and hemorrhage was intentionally provoked. The uvula and nodulus were elevated to expose the lingula, but these structures were not damaged. Following the operation these animals had no disturbance of either equilibrium or eye movements. The labyrinthine reflexes, posture and motor performances were not altered. Histologic examination of serial sections showed no damage to cerebellar and medullary structures with the exception of the uvula where some cortical degenerative areas were found (Fig. 8). These data are important because they demonstrate that even lesions of the uvula, in addition to surgical trauma to regions adjacent to the nodulus, fail to produce disequilibrium and abnormal eye movements.

The release of vestibular centers from cerebellar inhibition has been postulated to account for disturbances in equilibrium and eye movements,^{2,5,18} but the neural mechanism underlying this release phenomenon remains unknown. Spiegel and Scala⁵ found that after lesions of the lobulus posterior medianus (tuber vermis, pyramis, uvula and nodulus) in the cat, the severity of postural nystagmus was positively correlated with the duration of post-rotatory nystagmus. Both increased, then gradually decreased during the course of daily post-

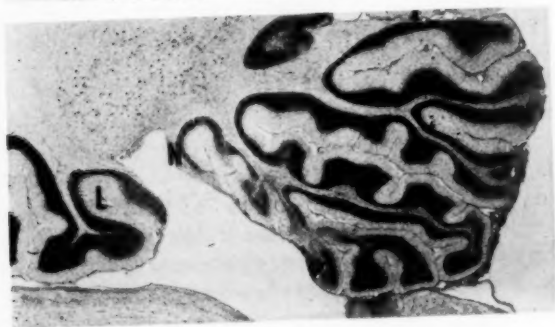
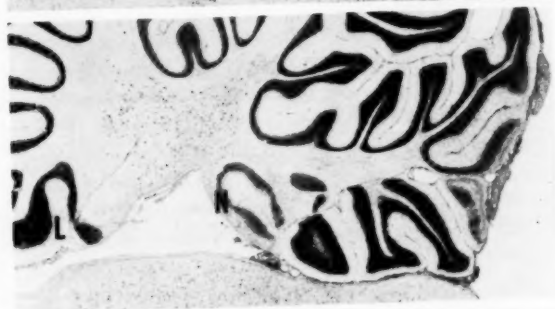
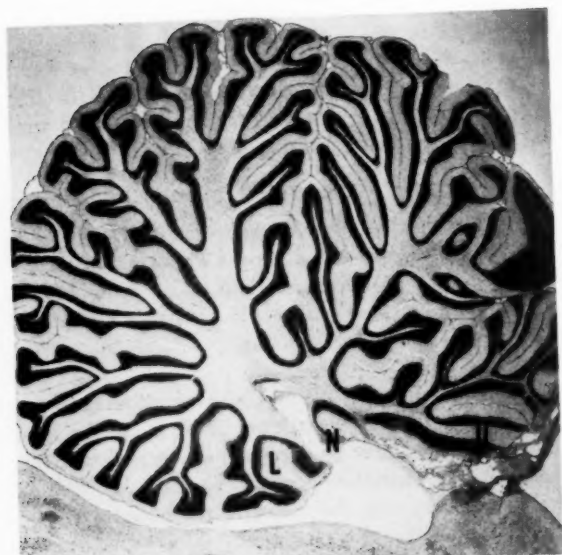


Fig. 5.—Cat A-456. Ablation of the nodulus. Midline section shows complete removal of the nodulus and half of the uvula. Cerebellar and vestibular nuclei, rhomboid fossa and cerebellar peduncles were not encroached upon. Animal sacrificed 38 days after surgery. Nissl's stain, X8. In this and following photomicrographs, structures are identified by the symbols: L, lingula; N, nodulus; U, uvula. To estimate the extension of the lesion in nodulus or uvula, or both, use the photomicrograph of Figure 8 as standard.

Fig. 6.—Cat A-456. The two lower photomicrographs illustrate that the nodulus was incompletely removed. The photomicrograph is a section lateral right to that presented in Figure 5, while the lower one is lateral left. Nissl's stain, X11.

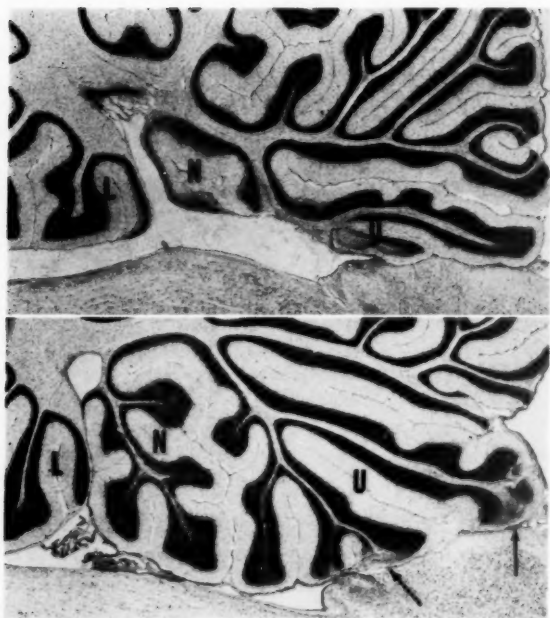


Fig. 7.—Cat A-457. Ablation of the nodulus. Animal sacrificed 36 days after ablation. The serial sections revealed that about half of the nodulus and part of the uvula were removed. The disturbances of both equilibrium and eye movements of this animal were mild. Nissl's stain, X12.

Fig. 8.—Cat A-461. Control experiment. The IV ventricle was opened, choroid plexus partially removed, and uvula and nodulus elevated until lingula was seen. These structures were not damaged. The photomicrograph shows no damage to the nodulus. The arrows point to a spotty cortical degeneration of the uvula. Animal sacrificed 30 days after operation. Nissl's stain, X12.

operative observations. Spiegel and Scala inferred from these data that the cerebellar operation produced a state of hyperexcitability of the vestibular nuclei which may explain the appearance of spontaneous or postural nystagmus, or both. In our series, neither spontaneous nystagmus nor prolonged postrotatory nystagmus were observed. The fact that Spiegel and Scala⁵ produced extensive cerebellar lesions may account for the discrepancy in results of the two experiments.

The finding that the direction of postural nystagmus was consistently in the vertical plane indicates that the signals from the

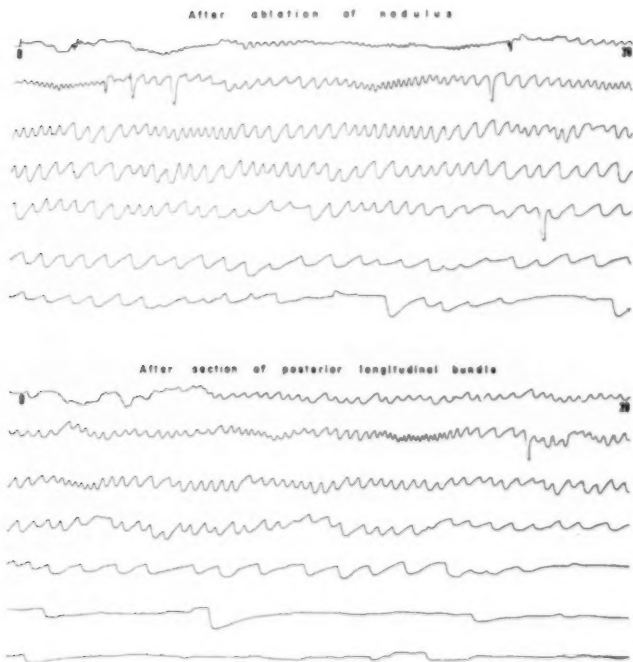


Fig. 9.—Cat A-674. Nystagmograph taken in hanging head position two days after removal of nodulus. The onset of postural nystagmus was delayed, nystagmic outbursts occurred from time to time and the total duration was about 140 seconds. The effect of sectioning the medial longitudinal fasciculus is presented in Figure 10.

Fig. 10.—Cat A-674. Effect of sectioning the medial longitudinal fasciculi upon postural nystagmus illustrated in Figure 9. The comparison reveals that only its duration was influenced; other characteristics were not modified.

vestibular receptors activate primarily the motoneurons of the third cranial nerve. If the trochlear or abducens nuclei were involved, then rotatory or horizontal nystagmus should be present. Horizontal nystagmus was never observed and rotatory nystagmus only occasionally. Some studies still in progress suggest that transmission of the signals to the oculomotor nuclei is through some pathway other than the medial longitudinal fasciculus. A deep transverse cut just below the inferior colliculi, performed two days after ablation of the nodulus, failed to modify the postural nystagmus (Figs. 9 and 10). An expla-

nation, admittedly speculative, is that release of vestibular centers from cerebellar inhibition permits convergence of peripheral signals toward the oculomotor nuclei presumably through a neural pathway within the reticular formation. There is anatomical evidence for this statement. Lorente de No¹⁹ described multiple pathways between vestibular nuclei, reticular formation and the motoneurons of cranial nerves to ocular muscles. He demonstrated that all the vestibular reflexes involving the ocular muscles were present after sectioning the medial longitudinal fasciculus and tractus vestibulo mesencephalicus. In view of these findings, he concluded that "the complicated vestibular arcs closed through the reticular substance are able to set up reflex reactions extremely similar to those produced by activity of the whole system."

To recapitulate, it has been postulated that the nodulus acts as an inhibitor of the vestibular centers. The question now arises as to whether the nodulus might also subserve other functions. Two studies suggest that it does. Dow³ found in monkeys that after recovery from bilateral labyrinthectomy, an asymmetrical lesion of the nodulus and uvula produced "imbalance of the reflex patterns of posture and movements." We repeated Dow's study in the cat; ablation of the nodulus two months after bilateral labyrinthectomy reproduced the disturbances in equilibrium seen in animals without deafferentation, but neither spontaneous nor postural nystagmus were observed. The function of the nodulus suggested by these experiments is that in addition to acting as an inhibitor of the vestibular centers, it subserves, in combination with other systems, the maintenance of equilibrium.

A striking feature of our experiment was the rapid compensation for the cerebellar deficiency. Disturbances of eye movements recovered in about 10 days and disequilibrium in 20 to 25 days. No attempt has been made yet to identify the structures that are responsible for compensation following ablation of the nodulus and uvula. Whether compensation is brought about by the cerebellum or higher centers is not known. The work of Walker and Botterell²⁰ indicate that, in the monkey, lesions of one half of the cerebellum are compensated by the remaining half. Aring and Fulton²¹ found, also in the monkey, that the frontal cortex, in particular the area 6, plays an important role in the compensation phenomenon. These investigations suggest that the mechanism may be a replacement of the function of one structure by another structure. Replacement seems to be the mechanism which compensates disturbances in eye movements, while a learning process may also be involved in compensation of disequilibrium. This is supported by the fact that disturbances in

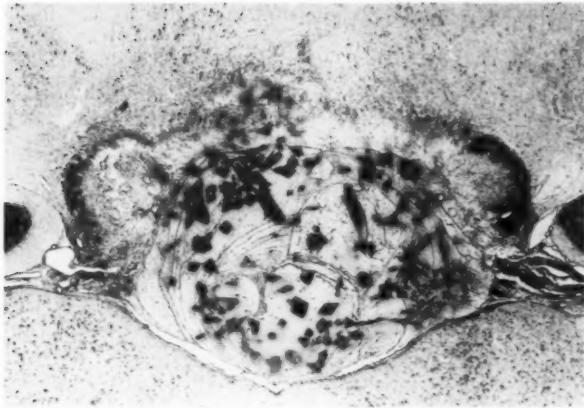


Fig. 11.—Cat A-569. Removal of the nodulus followed by bilateral cocainization of middle ears. The transverse section of cerebellum and brain stem shown in this photomicrograph revealed ablation of nodulus and also shows the IV ventricle occupied by a clot in process of organization. This clot probably caused an impairment of the general condition on the fifth postoperative day. The animal became paralyzed. Postural nystagmus was still present. The conditions of vestibular and cochlear receptors are illustrated in Figure 12. (Nissl's stain, XII).

motor performances can be demonstrated by appropriate tests in an apparently compensated animal.

As Botterell and Fulton²² pointed out, the deficiency following a localized cerebellar lesion in man does not parallel that in lower animals. The cerebellar organization differs largely from one species to another; as a matter of fact, even among subspecies there exists marked differences. Consequently, the practical application of ablation experiments or other procedures to human pathology should be used on a comparative basis, keeping in mind as stated by Botterell and Fulton "that the organization of human cerebellum differs widely from that of lower animals." Considering the difference in cerebellar organization between cat and man, we assume that in the latter lesions of the posterior paleocerebellum, which includes the nodulus, may be associated with disturbances of equilibrium and postural nystagmus. In such a case, the disability may be due to impairment of the dual function of the nodulus. It must be clearly understood that a cerebellar lesion that includes the nodulus is not always associated with disequilibrium and postural nystagmus. This is a well known

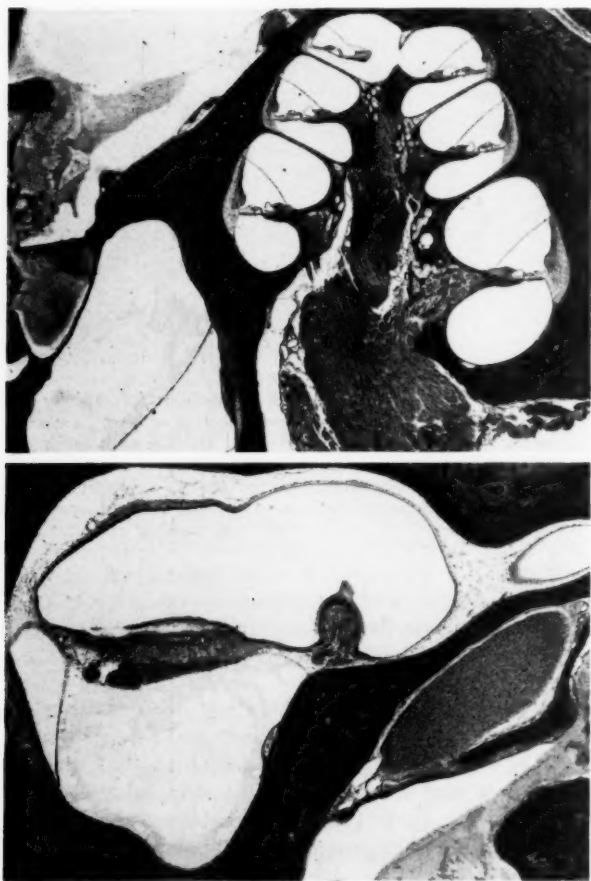


Fig. 12.—Cat A-569. These photomicrographs illustrate that cocaineization of the middle ear produced a temporary functional paralysis of vestibular receptors. The structures of both vestibular and cochlear receptors showed no abnormalities. (Upper photomicrograph, H-E, X31; lower, H-E, X21).



Fig.13.—Cat A-462. Ablation of the nodulus followed 24 hours later by bilateral labyrinthectomy. The photomicrograph shows an extensive lesion including nodulus, uvula and pyramis. The cerebellar nuclei probably were encroached upon. Arrow points to a piece of gelfoam attached to cortex of pyrami. The postural nystagmus following cerebellar lesion was permanently suppressed by bilateral labyrinthectomy. Animal sacrificed 45 days after removal of nodulus. (Nissl's stain, X14). The lesion in vestibular receptors is illustrated in Figure 14.

fact, which has been observed often in clinics. Similarly, disequilibrium and postural nystagmus may be produced by lesions other than those in the nodulus. The clinical value of these two signs, disequilibrium and postural nystagmus, is in conjunction with or absence of other signs.

II. EFFECTS OF DEAFFERENTATION OF VESTIBULAR CENTERS

Spiegel and Scala⁵ demonstrated that postural nystagmus which follows lesion of the lobus posterior medianus can be suppressed by subsequent, bilateral labyrinthectomy. The experiments indicate that

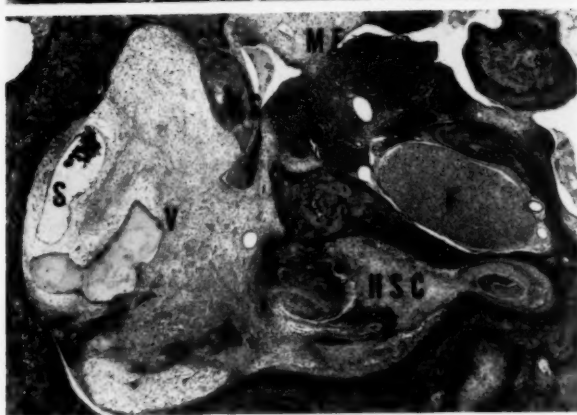
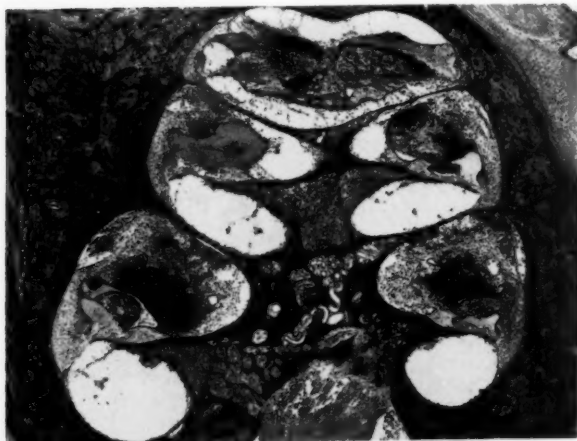


Fig. 14. (Upper and middle photomicrograph)—Cat A-462. Ablation of the nodulus followed two days later by bilateral labyrinthectomy. Upper photomicrograph shows degeneration of cochlear receptors. Fibrous and inflammatory tissues occupy middle ear and cochlea. Lower photomicrograph shows absence of vestibular receptors. Vestibule and semicircular canals are filled with fibrous tissue and new bone. V, vestibule. S, sacculus. HSC, horizontal semicircular canal. F, facial nerve. FS, footplate of stapes. ME, middle ear. (H-E, X25).

Fig. 15. (Lower photomicrograph)—Cat A-468. Ablation of the nodulus five days after bilateral labyrinthectomy. The photomicrograph shows an extensive degenerative process of the nodulus. The uvula also was encroached upon. (Nissl's stain, X13).

the presence of vestibular receptors is necessary for the occurrence of postural nystagmus; an opinion supported by the following series of experiments.

In four cats the nodulus was removed first and the animals were left to recover until disequilibrium and postural nystagmus were confirmed. Then 0.2 cc of a 10 per cent aqueous solution of cocaine was injected into the middle ears. The injection was done through the tympanic membrane with a 26 gauge hypodermic needle. Bilateral cocainization of the middle ears suppressed postural nystagmus and the classical syndrome of bilateral labyrinthectomy appeared for an interval of five to six hours. After this interval the disturbances of equilibrium and eye movements reappeared. The histologic damage of one case is illustrated in Figure 11. The serial sections revealed an extensive but incomplete removal of the nodulus. The uvula was partially damaged. The rhomboid fossa, cerebellar nuclei and cerebellar peduncles were not encroached upon. The IV ventricle was occupied by a large clot in process of organization. The cocainization of both ears did not produce detectable histologic damage in the vestibular or cochlear receptors as Figure 12 shows.

When bilateral labyrinthectomy was done during the period of cerebellar deficiency, postural nystagmus was permanently eliminated and the signs of disequilibrium were masked throughout the postoperative recovery by those of bilateral labyrinthectomy. Figure 13 shows the extent of the cerebellar lesion of one case in which nodulus, uvula and pyramis were encroached upon. The cerebellar nuclei might have been involved. This case exhibited the most extensive cerebellar lesion found in our series. The histologic study of both temporal bones showed otitis media, degeneration of the organ of Corti throughout the cochlea and proliferation of both fibrous and inflammatory tissues in the scalae vestibuli and media (Fig. 14). All vestibular receptors were absent, and the vestibule and semicircular canals were filled with fibrous tissue and new bone.

As one might expect from the experiments just described, ablation of the nodulus (Fig. 15) following bilateral labyrinthectomy in the acute stage failed to produce postural nystagmus and did not modify the signs of bilateral labyrinthectomy.

Comment: These experiments indicate that the stimuli that evoked the postural nystagmus after ablation of the nodulus originate in the vestibular receptors; postural nystagmus disappeared after bilateral labyrinthectomy or it did not appear when bilateral labyrinthectomy preceded ablation of the nodulus. The stimuli carrying the

information of position change are not altered by the cerebellar operation. What seems to be changed after removal of the nodulus is the neural mechanism of transmission within the central nervous system presumably brought about by release of vestibular centers from cerebellar inhibition. Since the mechanism of the vestibulo-ocular arc reflex is still obscure, the place where the cerebellar inhibitory action is effective remains uncertain. The afferent and efferent connections of the cerebellum^{1,19} suggest that the nodulus discharges upon the vestibular chains in the reticular substance but also directly upon the vestibular nuclei.

SUMMARY

This investigation supports the notion that the nodulus acts as an inhibitor of the vestibular centers. Disequilibrium and postural nystagmus in the vertical plane characterize the deficit following ablation of the nodulus. Unlike animals with lesions in the corpus cerebelli, no abnormalities in posture and no cerebellar ataxia are present. The occurrence of postural nystagmus requires the presence of signals from vestibular receptors indicating positional changes of the head, but disturbances of equilibrium do not. This indicates a dual function of the nodulus: inhibition of the vestibular centers and, in conjunction with other systems, maintenance of equilibrium.

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IX

MYOBLASTOMA OF THE LARYNX

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Myoblastoma of the larynx is a rare tumor. Recently on the broncho-esophagology service of the Jefferson Medical College Hospital we have had several patients with this unusual tumor of the larynx. The following presentation is a general discussion and review of the cases seen over the past ten years at the Jefferson Hospital.

The first reference to a myoblastoma in general was made by Weber¹ in 1854 when he reported a tumor of the tongue with microscopic features suggestive of myoblastoma. No references are made of this again until 1926 when Abriskoff² described a peculiar group of tumors which he believed arose from the primitive myoblasts following muscle injury. In 1934, Klemperer³ reviewed the world literature and was able to collect 50 cases of myoblastoma of all parts of the body. In this series, four cases originated in the larynx, and again in 1934 Kleinfeld⁴ reported on a single case of myoblastoma of the larynx. Since 1934, one finds in the literature reports of occasional cases, seldom numbering more than one, as reported by Geschlein,⁵ Bobbio,⁶ Freckner,⁷ Iglaue,⁸ Keohane,⁹ Crane,¹⁰ and Hinton and Weinberger.¹¹ The largest series of 14 laryngeal cases has been reported by McNaughton and Fraser¹² in 1954.

Although the tumors have occurred in all age groups, the greatest incidence has been in the third, fourth, and fifth decades. There does not appear to be any sex predilection. However, recently in McNaughton and Fraser's series of 14 cases reported in 1954, they reported tumor in nine males and four females. In the present report of five cases we have noted that four were males and one a female; thus, males appear to predominate.

Considerable speculation has persisted as to the etiology of the myoblastoma. Abriskoff in his original report suggested that their

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origin was associated with a degenerative process of muscle cells following trauma, but in a later paper suggested that certain lesions might have been associated with embryonic muscle cells on a dysontogenic basis. (The similarity of the myoblastic cell to regenerating muscle is striking. Godlewski¹³ described the fibrillar arrangement of the granules in these cells and suggested that the mature myofibrils might be formed as a result of the consolidation of these granules.)

It was suggested by Klinge¹⁴ that these tumors might originate from primitive myoblasts. Evidence to support this hypothesis was found in the similarity of the tumor cells and immature skeletal muscle fibers. Apart from histiocytes, striated muscle cells and myoblasts, the source of origin of granular cell myoblastoma has been attributed also to fibroblasts and Schwann cells of peripheral nerves.

Fust and Custer¹⁵ considered that the lesion was essentially a neurofibroma and Bangle,¹⁶ as a result of detailed histological and histochemical studies, stressed the relationship of granular cell myoblastoma to peripheral nerves. Pearse,¹⁷ also basing his conclusions on histochemical investigations, formed the opinion that the growth is a granular cell fibroblastoma in which a lipid containing substance similar to granules found in fibroblasts, is accumulated with the tumor cells; he believed the process producing such a change was one of degeneration.

Recently the subject of the genesis of these tumors has once again been open to question by the demonstration that tissue cultures of myoblastoma yield elongated spindle cells that bear a striking resemblance to young muscle cells. Their origin, then, to date, is unsettled but I believe most investigators feel these tumors arise as the result of misplaced primitive myoblasts.

Grossly, these tumors are generally small (0.5 to 2 cm) and sharply circumscribed. They may be encapsulated but instances of poorly defined lesions are also observed. The consistency of these tumors is usually firm. The cut surface of a myoblastoma varies in color from a gray yellow to a pearly white and may appear nodular or lobulated. The tumor is usually elevated above the mucous surface often resembling a pedunculated polyp.

Granular cell myoblastomas vary considerably microscopically. The characteristic elements are polygonal cells and ribbon-like syncytial masses. The nuclei are centrally located and the cytoplasm is free from lipoids, glycogen or amyloid. Cross or longitudinal stria-

tions are occasionally seen, and mitoses are usually absent. Not infrequently when these tumors occur subepithelially or submucosally the overlying epithelium undergoes striking hyperplasia known as pseudoepitheliomatous hyperplasia sometimes simulating the development of a frank carcinoma.

The majority of granular cell myoblastomas reported are histologically and clinically benign. The lesions are slow growing and local excision suffices in most cases for total cures. However, recurrence locally has been reported. Ravich, Stout, and Ravich¹⁸ feel that the malignant form of myoblastoma represented a polymorphous sarcoma. According to Howe and Warren,¹⁹ a myoblastoma should be considered malignant if the following criteria are met:

1. Atypisim of cells.
2. Excessive number of mitotic nuclei.
3. Spindle-celled sarcomatous pattern.
4. Local invasion.

In differential diagnosis one should consider other lesions simulating granular cell myoblastoma such as rhabdomyoma, sarcoma, epidermoid carcinoma, xanthoma, neurofibroma, and leiomyomas, papilloma, polyp, tuberculosis, and keratosis.

Since the lesion limited to the larynx appears to have benign characteristics treatment is local excision. If the patient is beyond the fifth decade frequent observation is indicated, and recurrence suggestive of malignant change should be treated accordingly with more radical surgical procedures. In general, with local excision the same principles of voice rest following removal of benign polyps is advocated.

Thus, in view of the comparative rarity of myoblastoma as a tumor type, the following five cases are being reported for the benefit of interested laryngologists and pathologists.

REPORT OF CASES

CASE 1. J.Z.W., a male aged 27, occupation paint sprayer, was admitted to Jefferson Hospital on January 30, 1950. His complaints on admission were hoarseness of six months' duration and a slight cough. Family history and medical history were non-revealing.

The patient smoked two packages of cigarettes daily and was a moderate user of alcohol.

On January 31, 1950, a direct laryngoscopy revealed a lesion on the left vocal cord. The lesion was described as a papilloma-like mass in the middle third of the left vocal cord. The cord had normal motility. This was removed and the histologic diagnosis was granular cell myoblastoma of the larynx. On February 2, 1950, a laryngofissure was performed under local anesthesia and the entire left vocal cord was removed. The pathologist reported that the specimen consisted of an irregular, pink, moderately firm piece of tissue from the larynx. The mucosal surface was smooth but the central portion was raised. The submucosa showed a nodular area measuring 5 mm in diameter beneath, and distinct from the mucosa. It did not seem encapsulated. The patient had an uneventful recovery and was discharged.

CASE 2. L.D., a white male, aged 53, occupation repairman. This man was admitted to the hospital on November 3, 1953, with a history of hoarseness of seven weeks' duration. There were no other symptoms of note. The family and medical history were non-contributory.

The patient smoked two packages of cigarettes daily and did not use alcohol. On November 3, 1953, a direct laryngoscopy revealed both vocal cords to be thickened and congested. On the edge of the left cord posteriorly there was a small rounded lesion which suggested a fibromatous process. This was locally excised.

Histological examination revealed that the specimen consisted of an irregular, elongated, grayish, hemorrhagic appearing tissue measuring 7 x 0.2 x 0.2 cm. It was moderately firm in consistency and was covered by a smooth white epithelial layer.

The diagnosis was granular cell myoblastoma of the larynx. The patient was discharged on November 5, 1953.

CASE 3. D.S., a white male, aged 24, occupation janitor. This man was admitted to the hospital on July 28, 1953, with a chief complaint of hoarseness of six months' duration. He had minimal associated dysphagia. Family history and past medical history were non-contributory. The patient smoked two packages of cigarettes daily, and did not use alcohol.

On July 29, 1953, a direct laryngoscopy revealed a cystic mass in the inner aspect of the right arytenoid. This was removed locally. Clinical impression at that time was a cyst of epithelial origin. The histologic report was as follows:

The specimen consisted of four, small irregular, moderately firm tan pieces of tissue, the largest measuring 1 x .3 x .2 cm.

The diagnosis was benign granular cell myoblastoma of the inter-arytenoid region. The patient was discharged on July 29, 1953.

CASE 4. J.G., a colored male, aged 32, occupation, ship fitter. This patient was admitted to the hospital on February 19, 1957, with a chief complaint of strangling cough of one week's duration. He attributed his cough to a chronic sinus condition. Family history and past medical history were non-contributory. On February 20, 1957, a direct laryngoscopy revealed a mass on the left arytenoid cartilage. This was removed with cupped forceps.

Histological diagnosis was granular cell myoblastoma of the larynx. The patient was discharged on February 21, 1957.

CASE 5. V.D., a white female, aged 45 years, occupation housewife. This patient was admitted to Jefferson Hospital on May 20, 1958, with a history of hoarseness of several months' duration. A direct laryngoscopy was performed on May 23, 1958, and a large polyp was noted on the left vocal cord and also on the posterior surface of the right cord. On May 23, 1958, the left cord was stripped with cupped forceps and the histological diagnosis of this tissue revealed a benign granular cell myoblastoma. Several days later tissue from the right cord was removed and the histological diagnosis was as follows: fibrous tissue partially lined by squamous epithelium compatible with a polyp of the vocal cord.

The patient was advised to continue with vocal rest for a period of one week and the last follow-up examination in October, 1958, revealed a normal functioning larynx with no evidence of polypoid changes on either vocal cord.

SUMMARY

Five cases of granular cell myoblastoma of the larynx are presented and discussed. Although the literature contains numerous references to this tumor, it is considered very rare in the larynx. To date most authors feel it is essentially a benign tumor. Local excision

followed by voice rest is the treatment of choice in lesions limited to the vocal cords.

Although granular cell myoblastoma is rare in the larynx, it warrants our attention in the differential diagnosis of tumors of the larynx, both clinically and microscopically.

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X

AUDITORY DISCRIMINATION AND
VISUAL PERCEPTION IN
GOOD AND POOR READERS

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Auditory discrimination and acuity as factors related to underachievement in school have received attention by investigators in the field of education. Programs to evaluate the hearing acuity in particular of school children are becoming increasingly more widespread, and are tending to encompass even the pre-first grade classes.

Although hearing acuity for pure tones is usually regarded as a good index by which to evaluate auditory capacity, and hence to rule out hearing as a reason for poor school achievement, nevertheless, the other important aspect of hearing, namely, auditory discrimination, perhaps deserves more attention than has hitherto been supposed. That the problem of auditory discrimination however has not been entirely neglected, is attested to by the fact that several diagnostic reading batteries, such as the one by Monroe,¹ include sections for assessing this dimension of hearing. In addition, reading programs at the elementary level as well as remedial reading procedures are constructed to provide training in learning to listen.

Bond² in 1935 compared good and poor readers in auditory acuity, discrimination and memory span. He found statistically significant differences in favor of the good readers in all three areas. Later, Kennedy³ investigated reading in relation to acuity for pure tones, but found no significant correlations except at the tenth grade level. She also studied auditory discrimination with the Seashore Pitch Test

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records and with nonsense syllables. In addition, the Western Electric 6-B pure tone audiometer was utilized in an attempt to measure the frequency difference limen. She concluded with reference to auditory discrimination that, "... the silent reading groups differ significantly as this factor was tested with the Seashore records on the 4-C audiometer." There were no differences, however, between good and poor readers on the frequency difference limen test.³

Robinson⁴ found acuity loss to be an infrequent cause of reading disability. Auditory discrimination and memory span were more frequently associated with a reading problem.

Henry⁵ showed significant differences in reading scores between subjects with normal hearing and those with high frequency losses.

More recently, Poling⁶ administered the Monroe-Sherman Group Diagnostic Reading Aptitude and Achievement Tests to 58 boys and 20 girls within the age range 8 to 13 years. This investigator did not find significant differences between good and poor readers either in auditory acuity for pure tones or auditory discrimination. In regard to auditory memory span, she concluded that it "... accounts for some of the failure in learning to read."⁶

It is apparent, therefore, that the research relative to auditory discrimination and reading ability is in conflict. That variables such as the influence of speech reading were not always adequately controlled was apparent from the review of the literature. Furthermore, test materials, varying in difficulty, could account for discrepancies in the results. It would appear, therefore, that further research is needed in the area of auditory discrimination and reading problems.

Reading is primarily a mental function. It is commonly mediated, however, through the visual channel. Obviously then, a disturbance of the sensory conducting mechanism resulting in seriously reduced visual acuity, or some other difficulty severely hindering impulse transmission would be expected to interfere with reading accomplishment. But, disregarding the cases of blindness or near blindness contingent upon acuity reduction, a myriad of investigations through the years have failed to uncover significant relationships between persistent reading disabilities and such deviations as esophoria, exophoria, hypermetropia, myopia, etc. Nevertheless, Poling⁶ has identified several factors which appear to be related to poor reading such as near-acuity and binaural vision. She emphasized the point, however, that her findings for selected subjects should not be generalized, but required further research on an unselected sample.

Thurston⁷ reported a factor analysis study of visual perception concerned with the identification of central rather than ocular factors. In addition to the major area of study, the battery of visual perceptual tests, such as various illusion tests, Hidden Pictures, Circles Test, Street Gestalt Completion, the Gottschaldt Figures, etc., was administered to a group of fast and slow readers at the college level.

Along with other results, Thurston reported a low positive correlation between reading rate and reading comprehension. He concluded, therefore, that his subjects could not be considered as either good or poor readers except as the terms related to speed of reading. In summarizing the results of the experiments with fast and slow readers, Thurston wrote:⁷

"Fast and slow readers should be studied carefully as to their perceptual differences in order to identify the perceptual functions which differentiate them. . . . It is quite likely that the purely ocular functions, such as accommodation and convergency, are marginal to this problem. Sensory acuity is also likely to be of secondary significance. With this admission, there has been a tendency to ignore completely the perceptual functions which can be distinguished from the more restricted ocular functions. By 'perceptual functions' we mean here the central processes that are initiated by sensory stimulation as distinguished from the physiological functions of the eye as such. . . . Too much attention has been probably given to the cultivation of reading interest and to personality adjustment in reading problems. The problem should be investigated more intensely in what seems to be the obvious direction."

The primary purpose, therefore, of this study was to investigate auditory discrimination and visual perception in good and poor readers, and to explore the relationship between such factors and reading achievement.

Specifically, the questions for study were as follows:

1. Do good and poor readers matched for I.Q., C.A., and sex show statistically significant differences in auditory discrimination as measured by the recorded W-22, the recorded Rush Hughes and the non-recorded Wepman tests?
2. Is there a statistically significant difference between the "difference scores" of good and poor readers? (The "difference score" refers to the magnitude of the difference between scores obtained on the

W-22 and the Rush Hughes tests. The concept will be discussed in the paper.)

3. Do good and poor readers, matched as described, show statistically significant differences on the Raven's Progressive Matrices, the Gottschaldt Figures, and a Figure-Ground test? (The Raven's Test is a nonverbal intelligence test. The Gottschaldt and the Figure-Ground are tests of visual perception.)

4. What are the relationships between some of the variables and reading?

SUBJECTS

Thirty male subjects ranging in age from 10 years 7 months to 12 years 9 months inclusive, were selected for study from a Kansas City, Missouri, public school. The experimental group consisted of 15 poor readers who were attending a reading clinic class. The control group of good readers were selected from the regular classes of the same school. The subjects were matched for chronological age within six months, and for Stanford-Binet I.Q., Form L, within 6 points. The I.Q. range for the 30 subjects was from 94 to 119. All were reported to have 20/20 vision as determined by the school nurse. Two of the control subjects wore glasses with correction to 20/20. The experimental group, in addition, had been referred for a complete ophthalmological examination. No visual defects were found. Specific socio-economic data were not collected on the subjects. The school, however, was located in a middle-income-group neighborhood, and teachers' estimates placed the subjects within the middle range.

EQUIPMENT

The recorded tests of auditory discrimination (W-22 and R. H.) were administered monaurally with a Beltone 12-A speech and pure tone audiometer. The assembly is portable. It consists of the audiometer, a matched pair of TD-39 earphones mounted in MX-41/AR cushions, and a turntable. The equipment was calibrated to National Bureau of Standard Norms for sound pressure, frequency and linearity of attenuators, and was checked periodically for sound pressure output with an Allison artificial ear, Model 3A.

The test chamber was an unoccupied storage room (20 x 20 ft.) on the second floor of the two story school building. The ambient noise level of the room during the tests did not exceed 50 db on the C-Scale of a General Radio Sound Level Meter, Model 1551-A. The

enrollment of the school was 250 students so that recess periods were at a minimum. Furthermore, no child was tested during recess. As the school was located a considerable distance from thoroughfares, relatively no traffic noise was experienced.

The tachistoscope employed in the study was manufactured by the Keystone View Company. Essentially, it consisted of an overhead projector and a flashmeter to control exposure time. The projector, as the name implies, is used to project 2x2-inch 35-mm projection slides on a screen. The Flashmeter provided exposure durations of 1/100, 1/50, 1/25, 1/10, 1/5, 1/2, 1 second and unlimited. A beaded motion picture screen, 50x50-inch, was located 11 feet from the subject's chair. The slides were projected on an identical area at the center of the screen during the tests.

THE TESTS

Auditory discrimination was measured with the Rush Hughes recordings of the Harvard PB (phonetically balanced) word lists, the C.I.D. W-22 records and the Wepman Test. The Rush Hughes and the W-22 tests (8) consist of 50 monosyllabic words in each list. The lists are balanced phonetically containing the sounds of the English language in approximately the same proportion as they would occur in a sample of ordinary speech. The W-22 lists, however, are much less difficult than the Rush Hughes Recordings. This is apparently due to distortion in the Rush Hughes recordings as well as the clipped and speedier method of presenting the words. In clinical practice discrimination scores obtained with the Rush Hughes records are about 20 per cent poorer than those obtained with the W-22 records. In scoring the tests a value of two points is credited for each word correctly heard. Since there are 50 words in each test-list, a score of 100 per cent would be credited if there were no errors.

Both tests (the W-22 and the Rush Hughes) are in general use in Hearing Clinics throughout this country, and represent the best available measures of general discrimination ability. The tests are administered at an intensity level sufficiently above threshold to assure a maximum score. Each test consists of several lists which are essentially equivalent as to difficulty. There are, therefore, several forms of the W-22 as well as of the Rush Hughes tests. For this study, the Rush Hughes recordings of test lists 7 and 8, and the W-22 recorded lists 1E and 2E were used.

The "difference score" or simply the difference between a subject's scores on the W-22 and on the Rush Hughes tests, is experimen-

tally employed in diagnosis in the Kansas University Hearing Clinic.⁹ As mentioned above, a subject will usually score 20 per cent higher in discrimination on the W-22 than on the Rush Hughes test. This difference score, however, appears to be relatively constant and independent either of degree of acuity loss or discrimination loss as indicated by the W-22 test. Clinically, we have observed that an abnormally large difference score is frequently associated with hearing dysfunction at the cortical level as indicated by an abnormal EEG and by neurological examination. The difference score is, therefore, of interest in this study.

The Wepman Test¹⁰ of auditory discrimination consists of 40 paired words. Thirty pairs are identical words and 10 pairs are different words. The subject, back turned to the examiner, is required to designate whether the paired words as spoken by the examiner are the same or different. In scoring the test, the number of right responses are recorded separately for pairs of words which are the same or different. In this study, however, correct responses from each category were totalled, and differences between groups analyzed from the composite scores.

The Raven's Progressive Matrices, 1938 version,¹¹ is an English nonverbal test of intelligence which is described by the author as: "... a test of a person's capacity at the time of the test to apprehend meaningless figures presented for his observation, see the relations between them, conceive the nature of the figure completing each system of relations presented, and, by so doing, develop a systematic method of reasoning."^{11,1}

The test consists of five sets of twelve problems. The initial problem in each set is practically self-evident with succeeding problems becoming progressively more difficult. There is no time limit for the test. Correlation with the Terman-Merrill Scale is reported to be .86. Norms for the group test on school children range from 8 to 14 years inclusive. There are also norms for adults. The scoring of the test is simply the number of problems correctly solved. Indices of mental capacity for children are determined by percentile points at half-year intervals. The test was of interest to us because of the extent to which visual perception might enter into the solution of the problems.

The Gottschaldt Test requires the subject to find a relatively simple geometric figure which is embedded within a more complex design. Teuber and Weinstein¹² have reported that brain-injured

subjects do poorly on the task. The test is divided into two parts, section A and B, with the latter containing the more difficult figures. The method and time limits for the test as described by Thurston⁷ were used. In scoring the test, the total number correct solutions for the two parts as well as in each part separately was utilized for statistical comparisons.

The Figure-Ground Test consisted of a series of nine slides of common concrete objects such as a cup, hat, penknife, boat, milk-bottle, bird, hand with pencil, iron and basket. The objects were imbedded in homogenous backgrounds of wavy lines and squares.¹³ The objects as well as the backgrounds were done with black India ink. The slides were projected on the screen by means of the tachistoscope for exposure durations of $1/5$, $1/2$, 1 second and unlimited time. The scoring involved the types of responses (whether foreground, background or mixed) for the series at each exposure rate. The test is utilized to measure foreground-background disturbance, particularly in brain-damaged subjects.

In conjunction with the preceding battery, the scores from the California Reading Test (Form X), which had been administered to the entire school system during the month of May, were made available to us. A composite score of reading comprehension and vocabulary was used by us in the statistical treatment of the data.

PROCEDURE

All testing was conducted in the previously described room during a six weeks' period in April and May. The total test time per subject was about one hour and a half excluding the reading test which was administered by the school system. The battery was administered individually (with the exception of the Raven's Test where groups not exceeding four were taken) during three sessions.

In the first session, the hearing acuity and auditory discrimination tests were completed. The order of presentation of the tests was: pure tone thresholds, the W-22, the Rush Hughes and the Wepman. For the monaural tests (pure tone, W-22 and Rush Hughes) the right and the left ears were alternately selected as the first test ear to counter-balance possible order effects.

The clinical ascending method was used to determine the pure tone threshold. The W-22 and the Rush Hughes tests were administered at a comfort level setting above threshold as determined during

a short practice session. The practice session which preceded each test in each ear consisted of having the subject listen to five practice words from a different recorded list to ascertain his comfort level as well as to accustom him to listening over earphones. For the W-22 test the comfort level was always 40 db or slightly greater above the average speech reception threshold for the equipment, and 50 db or slightly greater for the Rush Hughes test.

For the Wepman test (the non-recorded test) the subject sat at a distance of one meter from the experimenter with back turned to control the factor of lip-reading. After the practice session as provided in the manual, the experimenter clearly enunciated the paired words, monitoring his presentation level at about a 70 db level as measured by the C-Scale of the General Radio Sound Level Meter. Following the subject's response the next pair of words were spoken and so on until the test was completed.

In session two the Raven's Test was completed, in groups not exceeding four, following standard procedure.

In the final session, the subjects received the Gottschaldt Figures, and the Figure-Ground Test. The Gottschaldt Test was administered as described by Thurston.⁷ In the Figure-Ground Test the nine figure-ground slides were presented, following a practice session with five slides, the figures of which were on white backgrounds. The subject sat at a distance of 11 feet from the screen in a darkened room. A 25 watt desk lamp to the rear of the experimenter provided sufficient illumination to write the responses. The slides were presented only once at exposure rates of 1/5, 1/2, 1 second and unlimited time in that order. All slides were presented at the exposure rate of 1/5 second, then 1/2 second, etc. Responses were recorded verbatim by the examiner.

In the statistical analysis of the data, *t* tests, *F* tests for homogeneity of variance and product-moment correlations were computed.²²

RESULTS

The results of the pure tone threshold tests for the experimental group revealed normal hearing in both ears. As a matter of fact only two subjects deviated from the norm by as much as 15 db, and in both cases at only 4000 cps in one ear. A primary pre-requisite for a control subject had been normal hearing for pure tones. There-

TABLE I

MEANS, STANDARD DEVIATIONS AND RANGES OF CHRONOLOGICAL AGE, AND THE TEST SCORES FOR EXPERIMENTAL AND CONTROL GROUPS. ALSO SHOWN ARE MEAN DIFFERENCES BETWEEN GROUPS AND T TEST RESULTS

	EXPERIMENTAL GROUP			CONTROL GROUP			M/DIF.	T*
	M.	S.D.	RANGE	M.	S.D.	RANGE		
C.A.	140.92	6.71	153-127	140.60	7.80	155-130	.32	.33
I.Q.	104.46	7.27	117- 94	107.66	6.20	119- 97	3.22	1.25
Read.	124.52	10.54	140-109	162.28	8.61	178-150	37.76	10.37**
W-22	93.80	3.64	98- 88	94.27	1.81	97- 90	1.53	.56
R.H.	68.73	5.58	79- 60	75.13	5.19	84- 63	7.60	3.15**
D.S.	25.70	5.27	36- 15	19.20	4.74	32- 11	6.50	3.44**
Wep.	34.40	2.64	38- 28	37.67	1.25	40- 35	3.27	4.36**
Rav.	29.00	16.41	38- 18	41.31	10.63	55- 20	12.31	3.66**
Gott.	19.40	8.24	29- 6	25.93	8.67	38- 6	6.53	2.05
G.A.	11.20	5.48	20- 2	14.00	4.38	21- 3	2.80	1.78
G.B.	8.20	3.37	20- 3	11.93	4.24	20- 3	3.73	2.57*
F.G.	6.00	1.30	8- 3	6.27	.97	8- 5	.27	.63

C.A. in months

Reading in months of reading age *df. at .01 level = 2.763

**df. at .05 level = 2.048

fore, no subject was considered for the control group unless the threshold test had indicated normal hearing in each ear for the frequencies 500, 1000, 2000 and 4000 cps. Statistical analysis of the data showed that the two groups were not different in threshold sensitivity. Other preliminary statistics indicated that there were no statistically significant differences between ears either on the W-22 or the Rush Hughes discrimination tests. Therefore, the scores for the two ears were averaged, and the data treated accordingly.

In Table I are shown the basic statistical computations for the two groups as well as mean differences and the t test results of these differences. The F tests had indicated that the groups were homogeneous as to variance at the .05 level, and are therefore not recorded in the table.

Inspection of the last column of the table shows that the groups did not differ significantly either in Binet I. Q. or chronological age. That the two groups were highly dissimilar in reading ability is evident not only from the statistically significant t test, but from

the fact that there was no overlapping of ranges. It will be also noted from the last column that the Rush Hughes, Wepman and the difference score differentiated the groups at the .01 level of confidence. Conversely, the difference between the W-22 scores was non-significant. It will be recalled that the W-22 is an easier test of auditory discrimination than the Rush Hughes test. To determine the extent to which intelligence could be a factor in the results, product-moment correlations were computed between the W-22 and I.Q., the Rush Hughes and I.Q., the Wepman and I.Q. and the Difference Score and I.Q. for each group as well as for the combined groups. The correlations ranged from .08 to .15 and are statistically nonsignificant. Apparently, none of the discrimination tests is related to intelligence as measured by the Binet Scale at this age level.

Next, correlations were computed between the auditory discrimination tests. As might be expected, the W-22 and the Rush Hughes tests were significantly related at the .01 level, (r was .58). No relationship was found between either of these tests and the Wepman (r was .043; r was .169). It would appear from these results that different aspects of discriminatory ability are being measured by the PB lists. One wonders whether or not the Wepman test is related to auditory memory span.

Correlations were then computed between reading and auditory discrimination. Correlations of .564, -.545, .589 and .079 were obtained respectively between reading and the Wepman, the difference score, the Rush Hughes and the W-22. The first three correlations are significant at the .01 level, and the last is not significant. The negative relationship between reading and the difference score means that as the reading score improved the difference score decreased. There is, therefore, a low positive relationship between auditory discrimination and reading as measured by the Rush Hughes, the Wepman and the difference score tests. None of the relationships is sufficiently high to predict reading achievement from discrimination ability.

By referring to the last column of Table I again, it will be seen that the Raven's test significantly differentiated the groups (t was 3.66) at the .01 level. The t results for the total Gottschaldt (t was 2.05) and the figure-ground (t was .63)* were statistically non-

*The figure-ground t of .63 is for the exposure rate $1/5$ of a second. Since the t results for slower exposure rates were even smaller, they were not recorded in the table. Only foreground correct responses received a credit of one. There were only two background responses and two mixed responses for each group. Hence, the groups were homogenous as to their response patterns.

significant. As a next step *t* tests for differences between groups were computed separately for section A and B of the Gottschaldt. The former did not differentiate the groups (*t* was 1.78). The latter *t* test differentiated the groups at the .05 level. In other words, on the more difficult section B of the Gottschaldt test the good readers were superior in performance to the poor readers at the .05 level. Correlations were then computed between the total Gottschaldt and the Raven's test, the total Gottschaldt and Binet I.Q. and the total Gottschaldt and reading. The resulting correlations were respectively: .489, .305 and .262. Only the *r* between the Gottschaldt and the Raven's test, therefore, was significant at the .01 level of confidence. Although the ability to disembed a figure from a more complex design is inherent in the Raven's test, its presence is too weak to predict performance on the Raven's from the Gottschaldt test. That the ability is not related either to reading performance or I.Q. as measured by the Binet test, is indicated by the non-significant correlations.

Finally, the raw data for the Rush Hughes, the Wepman, the Raven's and reading were converted to *T* scores. A composite score for the two auditory discrimination tests and the Raven's test was correlated then with the reading test. The correlation obtained from this procedure was .731. In view of the small number of cases in the study as well as the nature of the data the computation of multiple correlations did not seem justified. Although generalizations as to the predictability of reading achievement from the above mentioned battery of tests should not be made from our selected data, nevertheless, the results appear to offer promise and hence, merit investigation on a larger unselected sample.

COMMENT

The results of this investigation offer confirmatory evidence for previous research which has indicated that poor readers are significantly inferior to good readers in auditory discrimination. A further finding of the study is that the discrimination inferiority of the poor readers is a function of the level of difficulty of the test, which is not, however, correlated with intelligence per se as measured by the Binet scale. The two auditory discrimination tests (the Rush Hughes and the Wepman) which differentiated the groups (the experimental and control groups) were not related, and thus appear to be measuring different aspects of auditory discrimination. Past research is in agreement that poor readers manifest a deficit in auditory memory span. Whether or not the Wepman Test reflects this particular facet of discrimination cannot be determined from the data.

As we have noted in this paper, the W-22 and the Rush Hughes tests are phonetically balanced monosyllabic word lists, which are employed extensively in hearing clinics to measure discrimination ability. Poor ability in auditory discrimination is a relatively common occurrence in individuals with hearing losses even after their acuity deficits have been overcome through amplification. Such disturbances in auditory discrimination are particularly apparent in cases of Ménière's disease which is of cochlear origin, of tumors of the VIII nerve, and of tumors of the temporal lobe of the brain. Decreased auditory discrimination is also a common observation in old people, even though their acuity losses for pure tones may be relatively inconsequential. In other words, decreased acuity as a result of perceptive deafness is usually accompanied by a decrease in ability to discriminate speech clearly. In old people, however, discrimination disability is frequently much greater than would be expected on the basis of their actual acuity loss for tones. That the poor discrimination in such cases is attributable at times to a reduction in the spiral ganglion cells and fibers of the VIII nerve appears well established, especially in view of the work of Schuknecht et al.¹⁴⁻¹⁷ That poor discrimination at other times is due to central factors is clear from the research of Bocca.^{18,19}

During the past year at the Hearing Clinic of the University of Kansas Medical Center research has been in progress as to whether or not auditory discrimination tests, varying as to level of difficulty, might be utilized to differentiate primary cortical discrimination dysfunction from dysfunction resulting either from auditory tract or from cochlear lesions. We have observed,⁹ for example, that old subjects with similar hearing loss for pure tones and similar W-22 scores not infrequently manifest marked dissimilarity on the Rush Hughes test. It was suggested by Goetzinger and Rousey⁹ that a large difference score between the tests might be indicative of primary auditory-cortical area dysfunction superimposed upon the normal difference between the tests. For many old subjects as well as young subjects reduced discrimination on the W-22 test, as a result of perceptive hearing impairment, is common. Their Rush Hughes scores are also reduced. The difference score, however, is usually about 20 to 24 per cent. When this difference score increases markedly, cortical dysfunction is suspected. By cortical dysfunction is meant a disturbance in the primary auditory perception areas of the temporal lobes. It does not imply a disruption in language formulation or in the processes associated with an auditory symbolic disturbance as in sensory aphasia. Furthermore, it does not refer to mental deterioration. At the present time we are attempting to collect supporting

data for the hypothesis of the difference score from EEG tracings. As with all research the results have not been without disappointment. However, the findings have been consistent enough to warrant continued pursuit of the technique.

Of particular interest has been the consistency with which a large difference score in one ear has been associated with an abnormal EEG in the contralateral hemisphere. Bocca^{18,19} has demonstrated an absence of summation for speech in cases of temporal lobe tumors in the ear contralateral to the tumor under certain conditions of binaural stimulation. In view of the fact that each ear is represented bilaterally in the brain, that pure tone tests of hemispherectomized patients show no difference in pure tone sensitivity between ears, that unilateral differences associated with the reception of speech do appear to occur in the contralateral ears of temporal lobe tumors, the importance of the auditory decussation fibers for discrimination and in particular, their terminal endings in the primary temporal-cortical areas, is suggested. We have had only one hemispherectomized patient (a child of ten years) among our patients. Although her pure tone threshold was normal in both ears, the difference score was 20 per cent larger in the ear contralateral to the removed hemisphere.

In terms of the present study, therefore, the statistically significant larger difference score for the poor readers might suggest a reduction in function at the primary auditory-cortical level.

As indicated in our results the good readers were significantly superior to the poor readers on the Raven's test. Visual perception as measured by the Figure-Ground test at the designated exposure rates did not differentiate the groups. The difference between the groups on section B of the Gottschaldt test was, however, significant at the .05 level of confidence, with the good readers evincing a superiority. A correlation of .489, significant at the .01 level, was found between the total Gottschaldt and the Raven's tests. Although a low positive relation appears to exist between the tests, nevertheless, it is clear that the inferior performance of the good readers involves much more than the ability measured by the Gottschaldt. Other tests of visual perception might provide more fruitful results.

Socio-economic status of the subjects had not been specifically investigated. Teacher estimates classified the subjects within average limits. That this is an important variable of the Raven's test has recently been demonstrated by Sperrazzo and Wilkins.²⁰ Another variable frequently mentioned is the effect of emotional disturbance

on the test. Kasper²¹ reported that the Raven's test did not consistently differentiate psychiatric adults from the norms. Although we have not included emotional disturbance as a major point of research in this paper, we administered the battery with the exception of the visual perception tests (the Gottschaldt and the figure-ground) to 12 boys and 3 girls in a school for emotionally disturbed children. The average score for the Raven's test was only slightly below the norm for their age. Furthermore, their auditory discrimination scores were not different from the control group of good readers. This study will be reported in a subsequent paper.

SUMMARY AND CONCLUSIONS

Fifteen poor readers from a public school reading clinic were matched with 15 good readers from the same school on the basis of Binet I.Q. and chronological age. All were males. The subjects were administered a battery of tests which included the W-22, the Rush Hughes and the Wepman auditory discrimination tests, the Raven's Progressive Matrices (1938), the Gottschaldt Figures, the Figure-Ground test and the California Reading test, Form X.

In terms of the questions posed for study, first as to whether the groups differed significantly in auditory discrimination; second, as to whether they differed significantly on the difference score; third, as to whether they differed significantly on the Raven's test and the Visual Perception tests; and fourth, as to the relationships between some of the variables and reading, the findings were as follows:

1. The W-22 test did not significantly differentiate the groups. The good readers were significantly superior to the poor readers at the .01 level on the Rush Hughes, the Wepman and the difference score. The latter findings support past research which indicates that good readers have better discrimination than poor readers. The former finding (W-22) points up the consideration of the level of difficulty of a test. In other words, a relatively easy discrimination test such as the W-22 does not differentiate good and poor readers. In addition to these findings no correlation was demonstrated between any of the auditory discrimination tests and Binet I.Q. A positive correlation of .58, significant at the .01 level was found between the W-22 and the Rush Hughes tests. Neither of these tests nor the difference score correlated significantly with the Wepman test. Apparently, the Wepman test measures a different aspect of auditory discrimination than the PB tests. The Rush Hughes and Wepman

tests correlated with reading at the .01 level. The respective correlations of .589 and .564 are too low for predictive purposes. The W-22 did not correlate with reading.

2. The statistically significant difference between groups on the difference score (.01 level) plus the lack of correlation between the difference score and intelligence possibly is suggestive of a primary auditory-cortical dysfunction in poor readers. Implications relative to this possibility were discussed.

3. The difference between groups on the Raven's test was significant at the .01 level of confidence. Neither of the visual perception tests differentiated the groups. When, however, the two sections of the Gottschaldt test were analyzed separately, section B, the more difficult part, differentiated the groups at the .05 level. The correlation between the total Gottschaldt test and reading was .305, a non-significant relationship. Positive correlations (.01 level) were found between the Gottschaldt and the Raven's tests, and between the Raven's tests and reading. When a T-score combination of the Rush Hughes, Wepman and Raven's tests was correlated with reading, a correlation of .731 was obtained. Generalizations based upon these correlations should not be made because of the nature of the data. The findings with a combination of tests appear promising, and suggest further research with a larger unselected sample as well as with other tests.

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XI

VARIABLES INVOLVED IN AUTOMATIC AUDIOMETRY

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Since Békésy¹ first published a description of his new audiometer in 1947 there has been an increasing application of the technique of automatic audiometry to various measures of auditory phenomena, as well as its diagnostic use in clinical audiology. The Békésy instrument consisted of a device which slowly changed the test tone from low to high frequency and provided for a continuous change in intensity under control of the listener. As soon as a tone was just audible, the listener pressed a button automatically decreasing the intensity; releasing the button as soon as the tone was no longer heard. These variations above and below the auditory threshold were graphically recorded as a function of frequency, the result being, in effect, a listener-obtained audiogram.

Békésy considered these tracings of variability about threshold a measure of the difference limen for intensity and gave specific illustrations of very narrow tracings (small variability) obtained from perceptively impaired ears. This aspect of the use of automatic audiometry to so differentiate perceptively involved hearing losses in terms of such threshold variability measures, that is, the presence or absence of the so-called recruitment of loudness phenomenon, has given considerable impetus to its application in diagnostic audiometry. This is especially so since the automatic threshold trace is supposedly not subject to relative comparison as the various binaural and monaural loudness balancing techniques. Thus it lends itself to a certain efficiency of administration, the controls of presentation being automatically regulated, making it somewhat impervious to any clinical naivete which may exist.

The advantages of automatic audiometry are manifest. The instructions are simple and easily understood. The listener task is not

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difficult and evidently is not affected by long listening sessions. Corso,² for example, reports no significant shifts in the mean threshold for 1000 cps between listening times of 5, 10, and 20 minutes in duration. And certainly the recording of a threshold audiogram uninfluenced by the examiner, except in the giving of the initial instructions, is most desirable.

SOURCES OF VARIATION - LISTENERS

For other considerations it seems reasonable to look at the listener response in terms of possible sources of variability. If we are to assume that certain types of abnormal ears are going to give, in addition to depressed thresholds, abnormally small measures of variability about threshold, the behavior measured must be sufficiently different from normal ear behavior to be detected. The closer the normal response is bracketed, the more sensitive the measure becomes. However, when we look at the reports of the excursion of normal traces we find that Lundborg⁵ cites between 6 and 9 db with extreme limits of 5 and 20 db, Békésy¹ indicates fluctuations of about 5 db as normal, and Reger⁶ reports 6 to 8 db as the normal zone of uncertainty about threshold. These reported norms obtain with an attenuation rate of 2 db per second. In the responses of seven relatively sophisticated normal hearing listeners, we measured ranges of from 4-9 db, 5-17 db, 8-15 db, and 10-30 db for attenuation rates of 1, 2, 3, and 6 db per second respectively. There definitely seem to be "narrow swingers" and "wide swingers" within the normal hearing population, the categorization dependent primarily on the criterion each listener sets up for a just audible tone. The wide swingers quite possibly wait for complete tonality and recognition while the narrow swingers are willing to respond to the initial "roughness" just before tonality. In any case, the narrow swinger who suffers an auditory impairment may give a false positive in terms of the presence of recruitment as measured by an automatic audiometer trace unless we are able to make a comparison to his trace before impairment or to his normal ear if the impairment is unilateral, as it often is in cases of Ménière's syndrome.

DIFFERENCE LIMEN VS. THRESHOLD VARIABILITY

Hirsh, Palva, and Goodman³ have emphasized that automatic audiometry is not a measure of the intensity difference limen but simply a measure of the variability about the absolute threshold.

When we compared such measures of variability for seven normal hearing listeners for three two-octave bands, 125-500 cps, 500-2000

cps, and 2000-8000 cps, we found that the mean variability about threshold for four attenuation rates, 1, 2, 3, and 6 db per second was consistently smaller for the lowest frequency band. This certainly does not agree with the increase in the size of the intensity difference limen as frequency is decreased; rather it would indicate that the traces are more closely related to loudness increase as Hirsh and his co-workers suggest. Such a relationship would give additional credence to a threshold variability measure as an indication of abnormal behavior to loudness changes.

ATTENUATION RATE

As the rate at which the intensity is changed increases, the excursions about threshold increase. Corso² reports a significant difference in such excursions occurring when the attenuation rate is increased from 1 db to 2 db per second. He concludes that this may be a consideration in the preference for the slower attenuation rate, indeed he suggests .5 db per second as preferable. However, other factors should be considered. When the tone appears and disappears relatively slowly, the subject may tend to become bored with the listening task and perhaps allow his attention to wander. With a relatively rapid attenuation rate, the listener is given no opportunity to "wait" for the tone to appear and disappear; he is too busy listening. If the increase in attenuation rate forces the listener to be more alert to the task at hand, it is reasonable to assume a threshold measure of greater validity should result. We found a tendency for lower thresholds, especially in the 4000-8000 cps band, as attenuation rate was increased from 1 db per second. The results cannot be considered conclusive but the trend is evident.

FREQUENCY TRAVERSE

The speed at which the frequency spectrum is traversed has a practical limit in that enough time must be allowed for the listener to display an adequate sample of excursions about threshold. If the traverse is excessively rapid and the slope of the hearing loss is steep, say greater than 20 db per octave, the subject may be constantly searching for threshold. Békésy's original instrument covered two decades (100 to 10,000 cps) in fifteen minutes. Because severe threshold drops per octave generally occur for frequencies above 1000 cps, important information is missed in this range when the frequency change is too rapid. We have found a faster rate than ten minutes is not desirable. A careful look at a specific frequency or a particular octave as a function of time is valuable but not always possible in

the clinical situation. If the frequency traverse is slow enough, an adequate listening sample at all frequencies may be obtained during the first run. The information thus obtained would suffice for diagnostic purposes.

DIRECTIONAL RECRUITMENT

The direction of the frequency change, whether from low to high or high to low frequency, is of importance in light of the findings of Hood⁴ regarding abnormal adaptation of the end-organ involved ear and both Reger and Kos⁷ and K. Schubert⁸ who demonstrated that temporary threshold shift occurs while threshold is being measured in perceptively impaired ears. From what we know of the fatigue effect, we would expect a smaller variability about threshold for such impaired ears if we measured threshold from a low to high frequency than if we reversed the procedure measuring from the high to the low. We tested a sample of 15 subjects, with perceptive hearing impairments and recruitment for frequencies above 1000 cps, over the octave of 2000-4000 cps. Two runs were done, one for ascending and one for descending frequency. In every instances the variability about threshold was smaller for the ascending condition than for the descending. In addition, the threshold at the midpoint of the octave was higher for the ascending than for the descending condition. Such directional recruitment measures, possible with the use of an automatic audiometer, are indicative of a heightened sensitivity for fatigue in ears demonstrating recruitment and may be the basis for a more precise measure of the degree of impairment relative to the amount of pathological involvement.

CONCLUSION

I have tried to point out and illustrate some of the factors and variables involved in the clinical application of automatic audiometry. The evidence indicates that it is a useful diagnostic tool in the evaluation of auditory impairment. There is no doubt its potential as such a diagnostic tool has yet to be fully explored.

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XII

CONTRIBUTION TO THE STUDY OF THE HISTOGENESIS OF THE AMPULLARY CUPOLA

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MÉXICO D.F.

The study of the auditive cupola and cristae from the morphologic and histogenic points of view is important because it permits comparison of the findings of physiologists with the data concerning the origin and shape of these organs.

We know that the processes which maintain the equilibrium of an organism in space still have many doubtful points. Among others, the fine structure and the function of the peripheral organ. Although numerous papers have been published on this subject, the incompleteness of the data and the often contradictory opinions are evident. For this reason we believe that any added observation may be of interest whenever a final synthesis on the matter is attempted.

The ampular cristae of the semicircular canals have been described by the majority of workers as conical prominent areas of connective tissue. On them lies an epithelium formed by three types of cells, the basal, the supporting and the ciliated, the latter being supplied with nerve endings. On the crista lies a mass of gelatin-like substance, the cupola, within which are embedded the cilia of the ciliated or sensitive cells.

This classical description does not, however, agree with the finding of Vilstrup.¹ This author studied the fetuses of an elasmobranch, *Acanthia vulgaris* (shark), and of codfish. He found in the developmental mechanisms and in the structure of these organs, different features than those described in other species. According to this author, the following takes place: In an early stage of development of the ear of the Acanthias, the epithelium of the crista is formed by two rows of superimposed cells. Some of these soon develop cytoplasmic prolongations which migrate in the shape of columns, into the lumen of the endolymphatic cavities. In so doing they carry with themselves

nuclear masses, usually elongated and picnotic in aspect. These protoplasmic columns become related to each other and form a syncytium whose elements arrange themselves for the greatest part, perpendicularly to the surface of the epithelium. They constitute thus, the fibers which traverse the cupola and give it a striated aspect. The protoplasms which migrate do not lose their connection with the cells which gave rise to them. They become thin and then acquire the aspect of cilia. These are the cilia described by the majority of the authors.²⁻⁹

Vilstrup adds the possibility that the protoplasmic columns and the nuclei forming a cupola shaped syncytium proceed from a previous caryokinesis which took place in the epithelial cells. This would mean that the material deposited in the endolymphatic space would be the content of some of the daughter cells.

The purpose of this paper is to investigate whether the histogenetic process of the cupola takes place in the chick embryos, by means of a mechanism comparable to that described by Vilstrup, in cartilaginous fish. Fowl are a zoological group which philogenetically are closer to mammals than fish, hence our interest in their study.

Material. Eighteen chick embryos were used (*Gallus domesticus*) of the following ages:

Age in days of incubation:	5	6	6.5	7	8	9	10	11	14
Number of specimens	1	2	1	2	4	2	4	1	1

Five-, six- and seven-day embryos were fixed *in toto* while the animal was still alive. The remainder were fixed immediately after beheading. The fixative employed was Bouin in the majority of the cases, excepting seven-, eight- and 10-day embryos. One of these was fixed with acetic sublimate. Paraffin inclusions were made. Harris' hematoxylin was used for staining and also eosin and hematoxylin of Delafield.

Findings. Embryos of five days' incubation: the otic vesicle is little advanced in its development but already one can see the subdivisions which will take place in the cavities of the internal ear. In the upper portion of this vesicle, precisely over the area where the nerve endings of the surrounding neurons terminate, one can see that the epithelium is thicker and shows abundant caryokinetic figures.

Six-day embryos are shown in Figure 1. The newly formed cristae are seen as an elevation of the connective tissue over which the epithelium is thicker and it shows a greater number of caryokinetic



Fig. 1.—Crista and cupola in a 6-day chick embryo.

figures than elsewhere. Over this prominent portion there is a seemingly denser substance because it stains more intensely than the rest of the contents of the ampulla. It has an affinity for eosin; its thickness is between 12 and 15 microns in its wider portion, generally the central portion, while in the narrower areas it measures from 2 to 3 microns. This condensation has the same shape at either side of the head. It shows small rounded granules more acidophile than the rest of the material. They are not exclusively in this area, but are also found in other cavities of the ear and in some blood vessels. Between the epithelium and the dense substance one finds in some cases a structureless space of from 6 to 7 microns apparently filled with endolymph.

In one six-and-a-half day incubation embryo the same findings were present as in 6-day incubation embryos.

In seven-day embryos (Fig. 2), the substance condensed over the cristae has a crescent shape, with its concavity facing the endo-

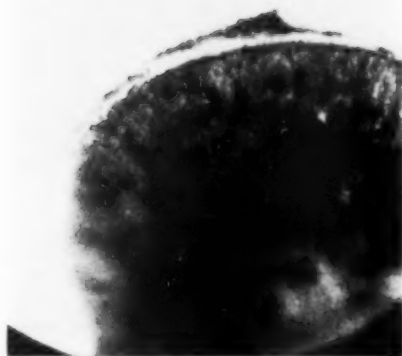


Fig. 2.—Crista in a seven-day chick embryo. Fine filaments are seen between the crista and the cupola. In the epithelium, a caryokinetic figure is visible.

lymph. This phenomenon is seen only in the most peripheral sections, while in the central sections of the crista, the crescent is parallel to it. The limits of the condensation are not precise; in some cases it is in contact with the epithelium. In two sections of one of the embryos, two or three filaments were found between the crescent and the crista; they were slightly granular, poorly defined and extremely thin. Caryokinesis in the epithelium are not so abundant as in younger embryos but when present the site of separation of the daughter cells is almost always perpendicular to the surface of the epithelium.

In eight-day embryos, the substance condensed over the epithelium is without precise limits; it is reticulated and contains abundant eosinophile granules. Some of the epithelial elements show a few thin protoplasmic prolongations resembling cilia, somewhat thicker at the base, a little curved and very short, measuring approximately as much as the small diameter of the nucleus of the same cells.

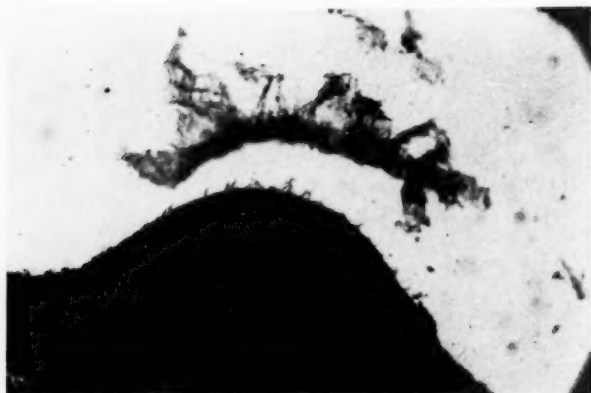


Fig. 3.—Ten-day chick embryo. Superexposed photograph with the purpose of making more apparent the cilia.

In nine-, ten- and eleven-day embryos (Fig. 3), the crista is conical and over it is the condensed substance corresponding to the cupola. This has a symmetrical configuration on both sides of the head and in the majority of cases there is a space measuring approximately 7 microns between it and the epithelium of the crista. The cilia are more evident and larger; they show a more marked acidophilia than does the material of which the cupola is made. The caryokinetic figures of the epithelial cells occur frequently even in 11-day embryos. The site of separation of the daughter cells is parallel to the surface of the crista.

Older embryos studied were 14 days old (Fig. 4). In some of them, the cupola presents striae parallel to the axle, that is, perpendicular to the surface of the epithelium; but in others the striae are rolled up in spiral fashion as described by Vilstrup. The cilia are markedly acidophile compared with the cupola. The cupola proper has a truncated distal end.

In the epithelium between the cells or within them, sometimes in the underlying connective tissue, granular formations are seen in the crista, which may be seen in seven-day-old and up to fourteen-day-old embryos. These granules are from two to four in number (Fig. 5); they stain intense purple with Harris' hematoxylin and measure approximately one-tenth the size of the nucleus of the epithelial cells. They occur infrequently having been found in only a few sections.

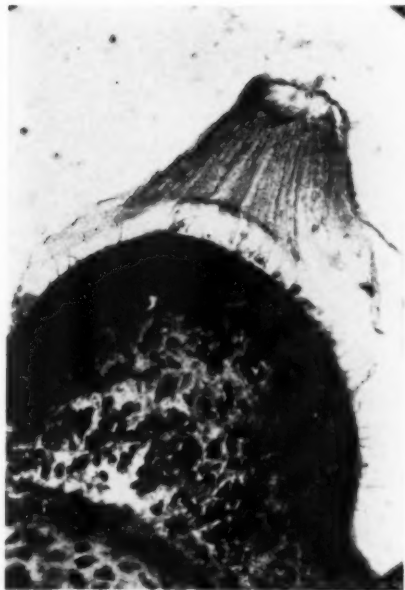


Fig. 4.—Cupola and crista in a 14-day chick embryo.

It is of interest that at the beginning of the formation of the cupola (six-day-old embryos) the cells of the crista are not yet differentiated into supporting and ciliated. This seems to substantiate Kolmer's view, who states that the cupola begins to appear before cellular differentiation takes place.

COMMENT

From about the middle of the nineteenth century Lang² described in the auditive cristae of the ear of Cypriniforms, cells provided with cilia. These prolongations were also found by Hasse³ studying fresh material in fish, reptiles and fowl. According to both authors the cilia are enclosed in fine channels which cross the jelly-like material of the cupola. Later, Kolmer⁴ supported Lang's and Hasse's views stating that similar observations had been made in human and other mammals' ears. Bowen⁵ regarded the cupola as a mere artifact due to the use of fixatives which condense the endolymph above the crista. However, he also described the cilia, some of which show motion, a

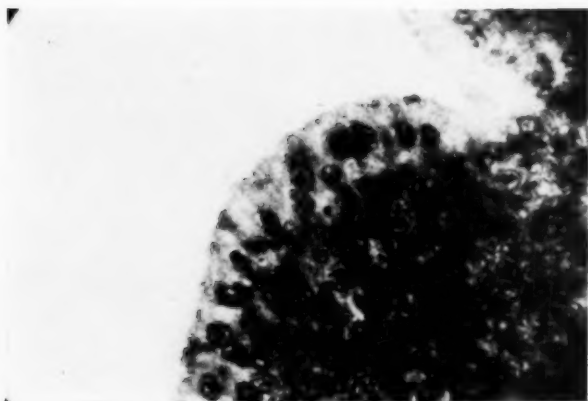


Fig. 5.—Granules in the epithelium of the crista of an 8-day chick embryo.

fact which led him to believe that there is no substance between them capable of restricting their activity. Wittmack⁶ believed that the cupola is formed by a secretion of the ectoplasm of the cilia and Shambaugh⁷ in studying new-born primates was able to observe the cilia enclosed in channels running perpendicular to the surface of the epithelium. Van der Strich⁸ believes that the cupola is a cuticular formation developed by underlying cells which form parallel prisms, united by cement, among which arise the sensitive hairs. Dohlman studied the mechanism and observed that the jelly-like substance reaches the opposite wall of the ampulla.⁹ Röthing and Brusch¹⁰ studied the sensitive epithelia in fowl.

Wersäll in 1956¹¹ studied the ear of guinea-pigs using the most advanced techniques of electronic microscopy. He made very careful observations of the auditive cristae and the cupola. His view is entirely opposite to that of Vilstrup. Wersäll points out that the sensitive cells are provided with true cilia which cannot in any way be considered as filamentous protoplasmic masses which have migrated from the sensitive epithelium. The cupola, according to Wersäll, is "a loose structure composed of numerous fibrils arranged in a network, whose more dense limit separates it from the endolymph."

These references are evidence that many authors agree in considering the prolongations of the sensitive cells in the category of cilia.

However, as mentioned, Vilstrup disagrees stating that they are merely protoplasmic columns which have migrated from the epithelial cells along with the nuclear materials.

In the chick embryos which were used for the present work, the cristae became apparent in six to seven day-old animals, which is in agreement with Herzog.¹² From this moment one can see on the epithelium a condensed substance near the cellular layer. We consider this condensation as belonging to the cupola and not as an artifact for two reasons: first, because it is present in the same shape and size on the homologous crista of the opposite side of the head and, second, because when we find dense masses in the endolymph in other areas of the vestibule, these have a very irregular shape and a variable localization and they are very inconstant.

The space between the base of the cupola and the sensitive epithelium is present in the majority of cases and it corresponds to the subcupolar space according to Vilstrup. In some preparations the most central sections show the cupola in contact with the cells, due probably to an artificial displacement, because it is not constant.

Columns of intracellular substance have not been seen to migrate from the epithelial cells to the endolymph; only rarely have there been found in the subcupolar space some fine and scarce fibers which do not look like Vilstrup's columns because of their dimensions, their number, their coloration and their relative positions.

Granules which stain intensely with hematoxylin have been seen at different levels within the thickness of the epithelium and in the underlying connective tissue but not in the endolymph. They are, furthermore, scarce and much smaller than the nuclei. They have also been found in areas of the epithelium quite distant from the cristae.

Cilia appear in chick embryos at eight to nine days' incubation. Two or three short prolongations are seen in each cell; they are thin from the very beginning, slightly thicker at the base than at the extremity. They do not seem to become continuous with the fibers of the cupola.

Shambaugh⁷ proved the absolute independence between the cilia and the striae of the cupola when he observed that they stain differently with Mallory's technique, that is, red oongo and hematoxylin. The material in the present observations has allowed us to confirm the

difference of staining properties between the cilia and the cupola fibers, a fact which leads us to believe that the latter are not the continuation of the former.

The caryokinetic divisions, quite numerous in the epithelium of the crista, on the whole follow a transverse orientation with respect to the surface, in such a way that the two daughter cells are aligned in a parallel fashion to the surface of the crista. If a caryokinesis were the stage previous to the migration of substances which will form the sincytium of the cupola as suggested by Vilstrup, it is probable that this division would take place in a plane parallel to the surface of the epithelium.

In some preparations and in the photomicrographs one notices among the cilia some globular bodies which stain lightly with eosin and never take the nuclear stains. These formations have been regarded by Wersäll and others as technical artifacts. It is not possible to regard them as picnotic nuclei as described by Vilstrup because they stain very weakly and not with nuclear stains; furthermore, they do not appear until the cilia are formed.

Because of all of this evidence one is led to believe that the formation of the cupola in chick embryos takes place without cellular migrations towards the endolymph as described by Vilstrup in *Acanthias*. Rather, one is led to believe that the cupola appears over the crista without any proof available as yet of a passage of substance from the epithelium to the cupola. This, on the other hand, increases gradually in size at the same time that its fibers organize. The subcupolar space remains untouched. In it appear the cilia which we regard as protoplasmic prolongations without nuclear content.

According to data derived from our material it seems unlikely that the fibrils of the cupola are the same as the cilia and that these might have acquired great length. We believe this especially because of the number of visible cilia in the subcupolar space is much smaller than the number of striae seen in the cupola proper.

With regard to the origin of the cupola, it is not possible with our material to propose any positive idea. It is possible that from a physical and chemical point of view one might clarify the problem in the future.

SUMMARY

A study of the histogenesis of the cupola in chick embryos was done in order to investigate whether in *Gallus domesticus* the process

described by Vilstrup takes place as he pointed out in an elasmobranch of the genus *Acanthias*.

In six- and seven-day-old chick embryos, the auditive cristae appear and over them, the incipient cupola, separated from the epithelium by a subcupolar space.

In seven- to fourteen-day-old chick embryos, the age at which the histogenesis of the cupola takes place, no columnar protoplasmic formations were seen in the subcupolar space nor elongated nuclei similar to those described by Vilstrup in *Acanthias*.

In eight- to nine-day-old chick embryos, protoplasmic prolongations appear which we regard in the category of cilia. They do not become continuous with the striae of the cupola and both have different staining affinities.

The phenomena of caryokinesis are frequently seen in the epithelium of the cristae, always in a perpendicular plane to the surface of the epithelium. For this reason it seems improbable that the cellular divisions are the previous stage of any protoplasmic migration towards the endolymphatic cavity.

The cupola begins to form before the differentiation of the epithelial cells takes place in the supporting and ciliated cells.

AV. CUAUHEMOC No. 300

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APRIL 13-14, 1959

XIII

THE WHISPERING SYNDROME OF HYSTERICAL DYSPHONIA

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In 1905 Freud published a case report¹ that was to startle and shock the lay world and, surprisingly enough, upset the medical world almost to a greater degree. This was the start of the psycho-analytical movement. The case reported was that of a girl (named "Dora" by Freud) who suffered from, among other things, hysterical aphonia. Today, this publication is considered a classic. It has been included in the Hutchins and Adler and the Encyclopedia Britannica "Great Books" series, and it is one from which all practicing physicians could benefit by studying.

The psychoneurotic manifestations of the voice vary from simply a noticeable alteration in pitch to a complete aphonia.² These may be syndromes of laryngitis, complete hysterical aphonia, a variety of speech defects (such as constant throat clearing, unconscious ejaculations, stereotypes and embolo-phrasias) and finally, a distinct clinical type of whispering dysphonia most commonly found in women. It is this latter group with which I am concerned in this study and I propose to report the course of their illness and the findings of their psycho-analysis. Often this type of patient is turned over to the psychiatrist and the actual psychic mechanism is never known to the laryngologist. I have, in twenty instances, followed with the psychiatrist these patients through their analysis in an effort to learn the psychic trauma and conversion neurosis involved.

CLINICAL DESCRIPTION

The patient typical of this whispering syndrome is a woman somewhere between puberty and menopause who comes to the laryngologist complaining of hoarseness. She is generally not particularly concerned with her plight. She is at first anxious to disclaim any nervous element in the condition, and the patient tells her story in a halting whisper which is obviously not hoarseness. In periods of

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relaxation, her voice may be nearly normal. Then the moment she becomes conscious of her difficulty again, the whispering is aggravated. At the first visit the patient is usually vague as to the exact onset and even more unwilling to speculate as to the cause.

Generally, these women are not too difficult to examine, in contrast to the anxiety neurotic. The vocal cords can be seen to move properly, approximate accurately, and are free of tumor or inflammation. In no instance have I found anything to suggest an "ephemeral adductor paralysis due to sudden anemia of the anterior cerebral cortex as in syncope" as described by Jackson.³

ETIOLOGY

Various pathological physiological explanations have been proposed for this whispering dysphonia, besides Jackson's. Furstenberg⁴ speculated on the possibility of a "disturbance in the nerve supply from the second motor areas of the cortex in this group of patients." However, the purely psychogenic causes described by Freud many years ago are clearly demonstrated in this series of cases.

There is evidence that this hysterical manifestation was even more common in primitive cultures than at present.⁵ Taking the Bible as an index, being "struck dumb" was apparently a fairly frequent reaction in man's earlier history, but the pressures of war more recently have demonstrated the same phenomenon in soldiers suffering from shell-shock. Therefore, the psychoneurotic mechanism is undoubtedly not related to any organic lesion.⁶ In the psychogenesis, the unbearable idea or conflict between opposing courses of conduct is rendered harmless by a process in which the sum of excitement is transformed into physical manifestations. This is what Freud called a conversion reaction, and it is important to recognize that it is not malingering.

Hysterical dysphonia is also to be contrasted with complete hysterical aphonia, in which the patient is subconsciously saying "I don't want to say anything." This latter is the reaction of the shell-shocked soldier who has witnessed an indescribably dreadful disaster. The neurotic female with hysterical dysphonia reserves a small channel for expression and is able to articulate in a voiceless whisper. The speech is most commonly affected because it is usually the first bridge between one person and another.

Psychiatrists today differentiate between an anxiety neurosis and conversion neurosis. The anxiety neurosis is based on a psycho-

REPORT OF CASES

HISTORY GIVEN LARYNGOLOGIST	PRECIPITATING CAUSE FOUND BY PSYCHIATRIST	PAST CAUSES FOUND BY PSYCHIATRIST
No. 1 Miss J.S., age 34, actress. Sudden onset of "hoarseness" night before consultation.	Mother saw partly-clad man leave daughter's apartment across the hall in early hours of the morning. Daughter said the friend forced his way into her bedroom and bed and that she could not call for help because of sudden hoarseness.	Three previous attacks of dysphonia apparently based on witnessing parents in intercourse as a child and promising brother "never to say a word." This episode had been repressed.
No. 2 Mrs. M.Y., age 42, TV and radio news commentator. Gradually increasing "hoarseness" for two weeks.	Patient had political ambitions and was an active political speaker. Had once been defeated for public office. Now husband was in what appeared to be successful campaign for county office. She "can't talk in his behalf," and unconsciously resents his success.	A marked Oedipus complex identifying herself with father who had been successful lawyer.
No. 3 Mrs. A.C., age 54, housewife. "Hoarseness" since husband died two weeks ago.	Guilt feelings over her husband's last words to her that she was "always hollering at me to move to another job that paid more money."	Complicated childhood background in which mother married many times. One stepfather was overly-attentive, giving many gifts. More than the normal physical contact with her.
No. 4 Mrs. A.D., age 46, wife of civic leader. "Hoarseness" since "just before our big party last night."	Patient former secretary of her 62 year old husband. He constantly talked of his first, now-deceased, wife, and what an accomplished hostess she was. On return from honeymoon he insisted patient give a large reception. She was fearful of her ability as a hostess with his old friends.	An Oedipal complex in which she identified her much older husband with her father.

- No. 5 Miss M.C., age 38, manager store. Much throat trouble in past; three "tonsillectomies."
- No. 6 Miss B.K., age 16, high-school student. "Hoarseness" of two days' duration.
- No. 7 Mrs. G.G., age 48, TV actress. "Hoarseness" of 1 week.
- No. 8 Mrs. G.B., age 32, "Hoarseness" of five days.
- No. 9 Mrs. M.Z., age 47, housewife. "Hoarseness" for 3 weeks.
- No. 10 Mrs. G.T., age 39, assistant office. "Hoarseness" 6 days.
- "Hoarseness" developed during violent argument with father over relationship with a male employee of store.
- Patient accidentally discovered masturbating; remained "sick and hoarse" until brought to doctor.
- Patient an alcoholic, had often used hoarseness to get out of TV shows when unable to memorize lines.
- Patient had been left to care for her seven small children without help, for four weeks, during which time she had not been out of the house once. When husband returned from business trip, he found her "barely able to speak."
- Patient severely scolded by husband for "talking too much and talking like an idiot" after drinks at a party.
- Patient found a used prophylactic in her husband's car after "business meeting."
- Oedipal complex, actual incestual relationship with father, founder of store who had turned management over to patient.
- Long-standing homosexual relationship with close friend.
- Psychopathic personality with long history of unsuccessful marriages, briefly successful acting jobs, etc.
- Schizoid neurosis with history of schizophrenia at age 20 when she was institutionalized and given shock therapy.
- Involutional depression. Had tearful episodes of self-reproach and obsessional behavior.
- Patient had never experienced a satisfactory sex life with her husband. There was an unconscious conflict between her homosexual feeling toward a female associate in her office and the sense of duty to her husband.

REPORT OF CASES (Continued)

HISTORY GIVEN LARYNGOLOGIST	PRECIPITATING CAUSE FOUND BY PSYCHIATRIST	PAST CAUSES FOUND BY PSYCHIATRIST
No. 11 Mrs. E.C., age 53, housewife. "Hoarseness" 3 days. Severe smoker's pharyngitis.	Husband brought a girl and another couple to his home for dinner prepared by patient. After dinner, football game. Patient drank brandy until she fell asleep intoxicated, awakening the next morning unable to speak.	Sexual relationship with husband never been happy. Because of religious taboos, had practiced <i>coitus interruptus</i> , leaving neither partner satisfied.
No. 12 Miss A.T., age 29, nurse. "Hoarseness," 2 weeks.	While bathing male patient, noticed him to develop erection.	Marked guilt feelings over phantasies and masturbation which she still occasionally experienced.
No. 13 Miss P.S., age 28, filing clerk. "Hoarseness" and non-productive cough, 2 days.	Defied mother and went out with boy she had recently met. Mother locked patient out. She spent night with female friend. When mother called to come home patient was unable to talk.	Marked Oedipus complex. Idealization of dead father and hatred for mother. Parents had unsatisfactory marriage.
No. 14 Mrs. B.S., age 30, housewife. "Hoarseness" of 3 months. Had consulted many throat specialists, brought in bottles of various tranquilizers, antibiotics, antispasmodics.	Favorite aunt with whom patient had identified herself suddenly found dying of cancer.	Had repressed memory of father's terminal illness from cancer of the throat when she was only five. Mother once told her not to mention cause of his death; "not to talk about such things and it's nobody's business!"
No. 15 Mrs. M.F., age 35, housewife. Many operations for questionable reasons. "Hoarseness" of 10 days.	Had married older wealthy man for whom she had no affection. She and mother lived with husband, who was away much of time. Patient fell in love with doctor; tried unsuccessfully to seduce him. Hoarseness appeared the next day.	Dominated by mother for years. Mother had forced her marriage because of personal interest in the man. Reproached her for her own "passions."

- No. 16 Mrs. A.P., age 48, divorcee. "Hoarseness" off and on for many months.
- No. 17 Mrs. A.K., age 48, housewife. Many episodes of globus hystericus and hysterical dysphonia. Previously refused to see a psychiatrist.
- No. 18 Mrs. T.C., age 28, divorcee. "Hoarseness," 10 days.
- No. 19 Mrs. B.B., age 39, divorcee. "Hoarseness" 2 days.
- No. 20 Mrs. M.F., age 37, housewife. Many operations for little reason. "Hoarseness," 2 months.
- Had gone to another city with man who promised to get a divorce and marry her. After two nights he disappeared and she came home "hoarse."
- Choked on a "hot-dog". Hoarseness and lump sensation persisted. Phallic symbolization?
- Experienced same nightly dreams in which man attempted to break into car with her locked inside. Finally "penetrated the door" and patient was unable to call for help.
- Choked by boy in fight. Insisted he had tried to kill her but did not want incident reported to police.
- Scolded by priest at confession for minor error. Unable to sleep that night, screamed suddenly, then "lost her voice."
- Marked bi-sexuality with "total inability to love." Patient had had both heterosexual and homosexual experiences by the age of 11.
- Had all the neurotic stigmata associated with oral eroticism — persistent thumb-sucking, oral phantasies, etc. Repressed them. Resultant guilt feelings and obsessional behavior.
- Childhood warnings from mother of "evil ways of men." Traumatic episode on honeymoon; husband became intoxicated and "sexually abusive."
- Pathological personality, long history of promiscuity. Troubles began age 13 when expelled from girl's boarding school for having man in room.
- Guilt feelings over childhood masturbation and phantasies; obsessional religious practices, went to church 2 or 3 times daily.

somatic symptom in which the patient has a demonstrable organic lesion. As an example, the young mother with a small laryngeal node from screaming at her children develops a cancer phobia concerning her hoarseness.

On the other hand, the conversion neurosis which is involved in hysterical dysphonia is an unconscious denial or repression of some experience by developing a physical symptom. There is no organic lesion and the woman is not only unconcerned, she almost seems relieved. There is typically a precipitating event or cause which the laryngologist may discover, but it is more important to realize that such a psychoneurotic reaction is evidence of a cause in the deep past which Freud contended was usually psychosexual.

MANAGEMENT

After taking a detailed history, a thorough physical examination of the head and neck is essential in order to establish confidence in the diagnosis. Besides the usual mirror examinations, there are two other helpful tests.

The first is Clerf and Braceland's "cough test"⁷ in which a few drops of any medication are instilled through the glottis into the trachea. This causes an immediate cough, the cords approximate, and occasionally, if taken by surprise, the patient will exclaim something in full and normal voice. This is more helpful in cases of complete aphonia.

The second test is a "cartilage manipulation test" which many physicians have used effectively in one form or another. Freud described a similar diagnostic device, as did MacMahan,⁸ although the latter apparently believed his manipulation to be therapeutic. In this procedure, the physician stands behind the seated patient and examines the neck as he would in palpating cervical nodes or the thyroid gland. Then the patient is told that her "laryngeal cartilages are going to be replaced through manipulation," so that she will talk normally while the physician "holds them in proper place." With this suggestion, many such patients will immediately speak out normally.

When the diagnosis is certain in the laryngologist's mind, the next question is: how does he get the patient into the hands of a psychotherapist? I almost never succeeded in doing so until I commenced the following procedure.

The patient is told without hesitation that I want Doctor — to see her immediately and that she should remain seated for a mo-

ment. No explanation is made of the nature of the physician's specialty (psychiatry). He is called to the office and into the examining room. (Fortunately, I have a few psychiatrists in my vicinity who are willing to come in such an emergency.) He is introduced and the patient acknowledges him in her hoarse voice. The cartilage manipulation is demonstrated to him. The psychiatrist is then left in the room with the patient and he attempts to establish a rapport with her to the point that she will go to his office.

When the psychiatrist has left, I return to the room and tell the patient that there is no other satisfactory cure—that I have tried drug therapy, narco-therapy, hypnosis, etc., without lasting benefit and that I believe that this type of treatment offers her a chance at a happiness she has never experienced.

A chart of twenty women in whom I was able to obtain a final psycho-analytical report is appended here. History and examination in each case suggested hysteria.

In other cases the patient discontinued her analysis after a few consultations. These are not included in this tabulation. Often the precipitating cause was immediately apparent to the laryngologist, but rarely was there an opportunity to gain insight into the deeper factors involved.

This is strictly an outline and does not pretend to be complete. All cases were not clearly defined, but some were mixtures of anxiety neurosis and other neurotic syndromes. Many of the patients were of high intelligence and successful in public life, others were of lower intellect and had lived an extremely confined domestic life.

SUMMARY

The patient with hysterical dysphonia needs psycho-analysis. Treatment by suggestion and hypnosis is only of transient benefit. The laryngologist must not scold or flatly accuse the patient of using this hoarseness for some psychological advantage nor should he fix the neurosis by treating it as if it were an organic lesion. The ailment must be taken seriously, however. A plan of management is described which has worked well in my hands. The patient is treated with kindness, reassurance, and encouragement and led into psycho-analysis directly. Finally, the laryngologist should not make the mistake of treating the patient "hoping that she will come around." This may re-enforce the hysteria and lead to dangerous amateur psychoanalysis.

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XIV

TRACHEOTOMY IN HEAD AND CHEST INJURIES

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The employment of tracheotomy for the removal of excess secretions from the lung in many surgical and medical conditions has greatly increased the indication for its use. With trauma now the third leading cause of death in the country, the use of tracheotomy in severe head and chest injury has become relatively commonplace.

Traumatic wet lung may be the result of any severe head and chest injury. It is probably seen most often when the body is thrown violently against the steering wheel or the dash board in automobile accidents. If this injury is not fatal at once, the clinical signs may appear to be relatively benign, and its true gravity may not be appreciated. If the increasing wetness of the lung that often follows these accidents is not relieved, more and more alveoli become blocked, and the patient may succumb to untreated or incorrectly treated asphyxia. This blocking of the alveoli produces changes in the blood chemistry. These changes are anoxemia, acidosis, increased serum potassium with low sodium and chloride levels. Thus asphyxia is the proper term to describe this condition. It should be stressed in dealing with head and chest injuries that severe asphyxia may be present without cyanosis, and that tracheotomy may be the only means of preventing a fatal outcome.

Most of the literature on this subject is found in the surgical, chest, and neurosurgical journals. However, when tracheotomy is used in the treatment of these conditions it becomes of primary importance to the otolaryngologist, because in many community and general hospitals his judgement will be relied upon as to if and when a tracheotomy should be done.

Let us look into the proper management of an accident involving several people who have sustained severe injuries. This might be the scene of an explosion, fire, tornado, train accident, and of course, by far the most common, the automobile accident. The obviously dead

and uninjured are passed over and attention is given to those injured but still living. The three things to look for immediately are asphyxia, hemorrhage, and shock. Hemorrhage and shock are outside the subject of this paper. As a rule, hemorrhage and shock do not kill as quickly as asphyxia. Usually impending asphyxia may be relieved by simply raising the jaw and pulling out the tongue. Blood clots and dental plates should be removed if found in the mouth or pharynx. This is particularly important in maxillofacial injuries. Oxygen may have to be administered on the way to the hospital. It should be stressed that if tracheotomy for traumatic wet lung is done it should be performed, if possible, in the hospital with proper help and adequate light.

Head and chest injuries affecting respiration to a dangerous degree are now more frequent in many hospitals than tracheal obstruction. The reasons for tracheotomy for obstruction of the glottis are obvious. The reasons for tracheotomy for obstruction of the alveoli, not as obvious, are just as important.

To produce cyanosis 5 gm% of reduced hemoglobin in the blood is necessary: therefore severe anoxemia may be present without any cyanosis. A patient who is anemic, say with 10 gm% of hemoglobin, could have half of his remaining blood supply unoxygenated without showing cyanosis.

The accumulation of high carbon dioxide tensions in the blood do not show color changes. A high carbon dioxide combining power plus an acid pH is necessary to prove respiratory acidosis because both a respiratory acidosis and a metabolic alkalosis produce high carbon dioxide combining powers. A direct pH should be determined when any doubt is present. The patient may die of asphyxia and never show more than a gray pallor to indicate the fatal chemical changes taking place in his blood.

Comroe and Botelho⁵ have shown that a group of competent observers could not recognize the existence of moderate arterial anoxemia when the presence or absence of cyanosis was the only sign employed.

Asphyxia in the central nervous system produces prominent symptoms. These are very similar to those produced by increased intracranial pressure. The observer sees central nervous system disturbances without localizing signs. The patient, if conscious, is restless and complains of needing more air. Serious symptoms are increas-

ing restlessness and anxiety. In a semicomatose patient there may be repeated rising up in bed, or later even thrashing about that requires the patient to have constant care. In some cases the excitement may take the form of mania. This was seen following the Coconut Grove fire in patients admitted to the Massachusetts General Hospital who had received damage to the lungs due to inhalation of hot gases.

These signs mean that the central nervous system is showing dysfunction from a combination of anoxemia, acidosis, hypercapnia, hyperpotassemia with low sodium and low chloride blood levels. Any depressing medications such as demoral, morphia, or barbiturates are contraindicated and may be fatal.

As these chemical changes in the blood are producing central nervous system damages that may cause death, an improved exchange of oxygen and carbon dioxide in the lungs is imperative. The use of oxygen by nasal catheter is quite inadequate. Acidosis and increasing serum potassium are not corrected and may be made worse by the drying and crusting effect of the oxygen and by attempts to exhale against positive pressure.

When it becomes necessary to remove excess fluids such as blood and mucus from the lower lungs, the human respiratory system is a difficult place in which to work, especially in the presence of head and chest injuries. There are protective reflexes in the pharynx and larynx that make it difficult to pass a catheter into the trachea for removing excess secretions even in a co-operative patient. This may become nearly impossible in a semicomatose patient who is struggling. In these cases it may be impossible to pass a catheter even through a direct laryngoscope. Bronchoscopy may be even more difficult or contraindicated in a patient suffering from head or chest injuries. Further, as more secretions accumulate, bronchoscopy may have to be repeated many times. An indwelling tube in the trachea is usually not tolerated by a patient partially asphyxiated and with a clouded mind. The body cannot store oxygen and it must be constantly supplied. At the same time carbon dioxide is accumulating in the blood, and when it reaches a level where the buffering mechanism can no longer function, there is a drop in the pH with an accompanying rise in serum potassium, and then cardiac standstill. Therefore it is obvious that secretions and blood from the chest must be removed to increase the exchange of gases in the alveoli.

The sensitivity of brain tissue to anoxia and the early degeneration that accompanies lack of oxygen to the cerebral tissues is well

known. In cerebral trauma a good airway and adequate oxygen are essential to good therapy. Tracheotomy offers the best means of increasing the oxygen supply to the injured brain tissues.

In any prolonged comatose state a tracheotomy may be required. It has been used successfully in diabetic coma, in coma secondary to a brain abscess, following a calcium embolus to the brain during a mitral commissurotomy, poliomyelitis, and for unmanageable secretions following a lobectomy or pneumonectomy for bronchiectasis, tuberculosis, or in cancer of the lung.

Tracheotomy is helpful in patients with severe head and chest injuries for the following reasons.

- 1) It reduces resistance to the flow of air into and out of the trachea by by-passing the narrow glottic chink and eliminating the resistance and turbulence of the pharynx.

- 2) It preserves the cough reflex while eliminating the bechic blast. The tussive squeeze is still intact. It is this tussive squeeze that pushes secretions into the larger bronchi where they may be reached with a catheter passed through the trachotomy tube.

- 3) It requires no co-operation or help from the patient. The airways may be kept free from secretions by a person without any unusual technical skill under the supervision of a competent nursing service.

- 4) It helps drain mediastinal emphysema of hemorrhage since the tracheotomy opening is directly connected with fascial planes which extend into the mediastinum.

- 5) It reduces the dead air space. The dead air space of the nose, mouth, pharynx, trachea and bronchi amount to about 150 cc. This is cut down by tracheotomy to roughly 50 cc. If a patient breathes 20 times a minute he is rebreathing 3000 cc a minute. After tracheotomy he is rebreathing only 1000 cc a minute. Thus 2000 cc of extra air or oxygen is given per minute. This may be life-saving in a patient with severely injured lung or brain tissue.

It should be pointed out that tracheotomy for severe head and chest injuries should be done when it is first thought that such a procedure may be necessary. Too frequently it is performed too late or not at all.

SUMMARY

Severe head and chest injury may produce respiratory difficulties which may prove fatal unless recognized early. Severe degrees of asphyxia may be present without cyanosis. Early tracheotomy in severe head and chest injuries is a valuable aid in treatment and may prevent a fatal outcome.

151 ROCK ST.

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CHRONIC HYPERPLASIA OF THE
MUCOUS MEMBRANE OF THE PHARYNX

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In 1901, A. Brown Kelly¹ described, for the first time, a clinical condition which he chose to call "sclerotic hyperplasia of the pharynx and nasopharynx." It existed in a 34-year old Scotsman who presented three striking features: 1) a greatly enlarged uvula, 2) very thick bands descending in each half of the posterior pharyngeal wall, 3) thickening of the roof of the nasopharynx making the lumen quite small. Histologically, a marked interstitial hyperplasia was seen. The diagnostic differentiation rested between rhinoscleroma, tertiary and hereditary syphilis.

Following Dr. Kelly's description, the following authors reported their observations of similar cases: Felix Semon² (2 cases, 1905), T. H. Halstead³ (1 case, 1908), A. L. Turner⁴ (2 cases, 1913), and H. B. Orton and P. Goldsmith⁵ (2 cases, 1933).

The attention of the author was attracted to this condition after observing the hypopharynx and upper esophagus of A.G., a man of 55, who complained of persistent dryness and fullness in the throat for ten weeks. On examination, the mucous membrane of the pharynx showed a very waxy pallor; the lateral pharyngeal bands were markedly enlarged, and a sausage-shaped epiglottis was observed. The uvula was absent, having been removed at the time of adenotonsillectomy at the age of 11 years. He vaguely remembers that at that time he was told that the uvula was large and obstructive. At examination the aryepiglottic folds and the arytenoids were so thickened that the mirror observation of the vocal cords was impossible. He was admitted to the Massachusetts Eye and Ear Infirmary on January 19, 1956, and x-ray studies of the chest showed bilateral apical tuberculosis. Biopsy specimens of the arytenoid, epiglottis and pharynx showed characteristic tubercles. The sputum was positive for tubercle bacillus. The patient was placed under active therapy with streptomycin and iontazamid. He was last examined in November, 1958, and had gained 23 pounds since he was first seen.

Roentgen examination of the chest in November, 1958, showed no active pulmonary disease.

Figures 1 and 2 show the roentgen appearance of the marked swelling of the structures above described.

Dr. A. S. MacMillan, the roentgenologist, in observing this, was impressed with the striking similarity to the roentgen study (Fig. 3) of M.B., a 55-year old white single female who was admitted to the Massachusetts Eye and Ear Infirmary on July 6, 1954, under the care of Dr. Philip Mysel.

This woman complained of a sense of fullness in the throat. It had become progressively worse for two years. She also complained of mild cough and had nocturnal noisy breathing. There was occasional blood streaking of the sputum, but no loss of weight or appetite. There was a past history of typhoid fever in childhood while resident in England, and erysipelas in 1924. Two siblings were living and healthy.

Her pharynx showed thickened, raised bands extending into the hypopharynx and a thickened uvula bounded laterally by atrophic palatine tonsils. The epiglottis was thick and the upper edge blunted by a marked diffuse swelling of a reddish purple color. The interior of the larynx could not be seen by indirect laryngoscopy because of the swollen aryepiglottic folds and arytenoids. The latter were uniformly thickened and the mucosa was not ulcerated. Direct laryngoscopy showed normal vocal cords. The general physical examination revealed aortic regurgitation because of a calcareous change in the aortic ring. Two serologic tests were negative; W. B. C., 6,500; B. M. R. + 8; fasting blood sugar - 101 mgms per cent; N. P. N. - 26 mgms per cent. Urine samples were negative for sugar and albumen. The temperature was normal during a nine-day hospital stay. Figures 4 and 5 show the microscopic appearance of a specimen of mucous membrane taken from the posterior surface of the right arytenoid.

Dr. Tracy Mallory, the pathologist, described "edema in the microscopic section, the edema being caused by many dilated lymph channels. There was localized infiltration of plasma cells and lymphocytes, but these do not tend to surround the dilated lymph vessels or account for the passive congestion." Figure 6 illustrates the swelling of the lower pharyngeal structures.



Fig. 1.—Roentgenogram showing marked thickening of the retropharyngeal wall, the epiglottis and the arytenoids in patient A.G.

COMMENT

One of Turner's cases was followed from 1903 through 1913. This case was discussed by Sir Felix Semon in his presentation of chronic hyperplasia of the mucosa of the pharynx in 1905.² He discarded the above case as well as several others because of the clinically obvious toxic and septic nature of their clinical course and because of their prompt resolution after specific types of therapy, such as in my case of pharyngeal tuberculosis.

It was, however, implied by Semon and emphasized by Turner⁴ that the possibility of an elusive septic origin must not be lost sight of in all these cases.

Actually, one of Turner's patients had a tracheotomy shortly after a sore throat. Turner suspected that the edematous hyperplasia



Fig. 2.—Roentgenogram showing the barium passing through the pharynx with difficulty. A large amount collects in the valleculae in patient A.G.

may have antedated the sore throat because the patient had the condition for the next ten years, during which time a tracheotomy was almost necessary for a second time because of a superimposed acute septic condition.

Turner stated that when one considers the frequency with which the throat is subject to septic infection and the rarity with which the condition we are considering is found, it is difficult to associate its origins with a septic background.

In my study of the reported cases, it is perhaps of more than coincidental importance that in two cases, of the seven (30%), Halstead's and the present one reported, a definite past history of erysipelas existed. It is noteworthy that the pathologist, examining the microscopic sections from Orton's patient in whom there was no history of erysipelas, reported lymph vessel occlusion with plasma



Fig. 3.—Roentgenogram showing marked retropharyngeal edema and edema of the epiglottis and arytenoids in patient M.B.

cells and lymphocytes. Mallory's pathological report in the case herewith presented indicates dilated lymph channels. This condition was also present in the first case, reported in 1901 by A. Brown Kelly, and also in one of Turner's cases. The described sparsity of polymorphomononuclear cells and the peculiar abnormality of the lymph channels in the microscopic descriptions of several of the reported cases suggests the possibility of a chronic nonseptic lymphedema of these tissues. There is strong microscopic support that this condition being described is a peculiar sequel of a specific streptococcal infection, with "waxy edema" the end result.

It is particularly impressive that in the reports presented, the vocal cords were not at all involved. The view of them was generally obstructed by supraglottic swelling, but when a glimpse of them was obtained, generally only by direct laryngoscopy, no alterations in the appearance of the cordal mucosa were noted. In keeping with

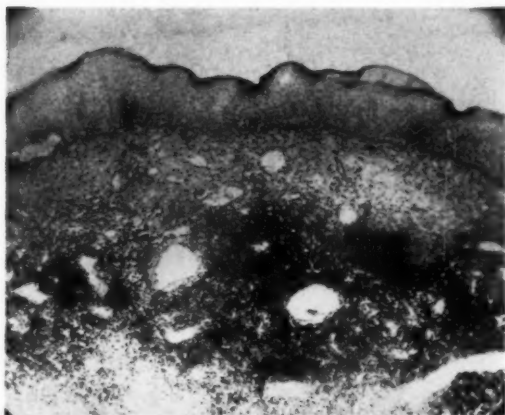


Fig. 4.—Photomicrograph of the mucous membrane, posterior surface of the right arytenoid, x 100.

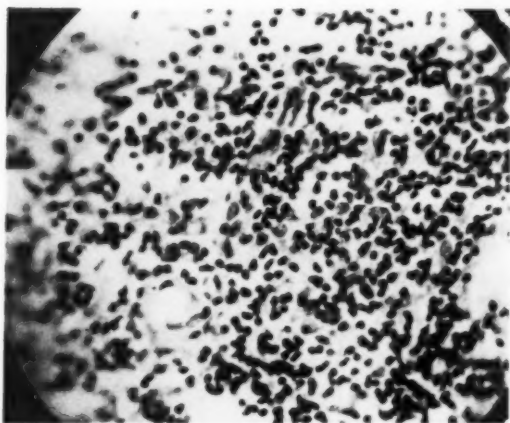


Fig. 5.—Photomicrograph of the mucous membrane, posterior surface of the right arytenoid in patient M.B., x 400.



Fig. 6.—Roentgenogram showing swelling of hypopharyngeal wall and arytenoids in patient M.B.

this, the clinical observation was repeatedly made that the vocal function in all the patients was undisturbed. The abrupt demarcation of the edema at the lower edge of the aryepiglottic fold in all these cases also suggests static lymphedema. The pooling and stasis of lymph flow could explain the swelling of the normal loose vascular fatty tissue of the ventricular folds to a very boggy state. This loose submucosa ceases abruptly at the vocal cords. It therefore seems that the likelihood of congestion due to stasis rather than to any spreading toxic surface infection is present in the condition. I am grateful to Dr. Philip Mysel for permission to report this case.

SUMMARY

1. The ninth case of chronic hyperplasia of the mucous membrane of the pharynx is presented.
2. A striking similarity in roentgen appearance has been demonstrated in a patient with tuberculosis of the pharynx.

3. There is a strong suspicion that this condition may be an end result of an alteration of lymph vessel function secondary to erysipelas.

285 COMMONWEALTH AVE.

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XVI

THE TEMPORAL BONE IN PAGET'S DISEASE

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Osteitis deformans or Paget's disease has been recognized with progressively increasing frequency since the first description by Sir James Paget in 1877. The number of papers dealing with the generalized disease has increased tremendously as there have appeared improvements in radiologic, biochemical and histopathologic techniques.

Deafness was noted in some of the early patients studied for Paget's disease. However, otologic investigation did not progress until the work of Otto Mayer which included histologic examination of the temporal bone of a patient with Paget's disease along with the clinical case and hearing tests. Further histopathological studies were carried out by the same and other authors, including Nager in 1919, and Jenkins in 1923 and 1928. There have since been further studies reported by Nager and Max Meyer, Brunner and Weber. Detailed descriptions of the temporal bones were reported by Wilson and Anson¹ in 1936 and by Tamari⁵ in 1942.

The present paper will present a case of Paget's disease which was followed at the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital for ten years and whose temporal bone was available for histologic examination.

Paget's disease is a chronic progressive disease which begins in middle life or later. The rate of progress is variable and may continue for many years. The disease may involve one, a few, or many bones and produces symptoms due to the deformity produced in the bone. The most frequently affected bones are the sacrum and the vertebrae, the femur, the skull, the sternum, and the pelvis. The diseased bones become enlarged and when these are weight-bearing bones, they become curved. The skull becomes large and misshapen so that there is progressive increase in the size of the head. This increase involves mostly the outer table, and the inner table remains comparatively unaltered. The base of the skull is sometimes involved and the pituitary fossa may be altered. The petrous part of the temporal bone

may also be involved and produce symptoms. The bones of the facial skeleton are less frequently involved than the cranium. If they are involved, the thickening and deformation lead a characteristic disfiguration of the face.

SYMPTOMS

For the most part, early diagnosis is difficult but becomes obvious when the characteristic symptoms appear. These are: the increase in circumference of the skull, the curving of the bones of the extremities, and occasionally, spontaneous fracture. Otologic symptoms are relatively infrequently seen unless the disease is far advanced. Barth in 1934 found deafness in 14 of 28 cases. He found that these were perceptive deafness in six and mixed deafness in eight. He, at that time, found that vestibular symptoms were more prominent than hearing loss in these cases. In 1936, Lindsay and Pearlman⁴ reported on 26 patients with Pagets' disease. Of these, eight showed involvement of the skull and of these eight, five had impairment of hearing. Although the hearing loss was most commonly perceptive, there was also a conductive loss which was marked in two patients and which was present in another patient during one stage of the disease. None of these patients had vestibular symptoms. The caloric response is usually normal or slightly decreased.

ETIOLOGY AND HISTOPATHOLOGY

Attention has been repeatedly drawn to the resemblance between Paget's disease and otosclerosis. Some have even concluded that otosclerosis was a retarded form of Paget's disease. The etiology for both diseases is still unknown although several theories have been advanced and then discarded, including inflammation and trauma; recently endocrine factors have been investigated.

The histologic changes in Paget's disease occur in the bone marrow and in the bone tissue itself. There is a combination of bone destruction and repair which leads to enlargement of the bone but not always an increase in the volume of bone tissue. Changes in the marrow lead to fibrosis. In Paget's disease there is considerable variability of the bone due to three factors. The first is the variable ratio of osteoclastic and osteoblastic activity in an area. Second, the variable speed of destruction and reconstruction. Third, the frequency of local remission followed eventually by resumption of activity.

The resorption by osteoclasts proceeds for some time and is followed by bone apposition by osteoblasts. These sequences of

destruction and repair are followed repeatedly in the same area and eventually lead to the development of mosaic bone, one of the characteristic features of Paget's disease. This, in effect, has the appearance of small pieces of a complicated jig-saw puzzle.

An active area contains numerous osteoclasts and osteoblasts. The marrow is always fibrous. Frequently, both osteoblasts and osteoclasts can be found on the same bone trabeculae. When there is marked osteoblastic activity, the trabeculae are often covered by a layer of osteoid. In an inactive area or one in remission the bone is found to be aplastic; that is, there is neither osteoblastic or osteoclastic activity. The marrow may become very fatty, and mature lamellated bone is laid down at this time. Sooner or later, however, the inactive period comes to an end and recurrence begins. This is characterized by a tunnelling resorption of the bone trabeculae removing the central part first. The repetition of the process leads to the gradual destruction and replacement of compact bone by spongy bone which is weakened. Thickening of the bone results from formation of bone trabeculae on the outer surface.

REPORT OF A CASE

Mrs. M. S., No. 408876: The patient was an 81 year old white female. She was first seen in the Out-patient Department of the Massachusetts Eye and Ear Infirmary in June, 1943, because of progressive hearing loss of many years' duration and a feeling of pressure in the ears. The drums at that time were described as being thickened, the Weber lateralized to the right ear, the Rinne with the 512 fork was 19/18 in the right ear and 28/20 in the left ear. Conversational voice was heard at one foot in the right ear and was heard at mouth to ear in the left ear. Audiogram showed a perceptive-type curve for both ears with the average loss in the left ear at 40 decibels and for the right ear at 60 decibels in the speech frequencies. She was given a nasal spray and politzerized, and with this treatment seemed to get along satisfactorily. A hearing aid was recommended but the patient found that she could get along fairly well without it. Over the course of the next six and one half years she was treated by her own physician for hypertension and by the Eye Clinic for glaucoma. In August, 1950, she was admitted into the emergency ward of the Massachusetts General Hospital because of a two-week history of light-headedness, floating sensation, tinnitus in the right ear, but no true vertigo. Her positive physical findings at that time included blood pressure 260/100. The head showed bony enlargement of the cranium in the right temporo-parietal region. The

temporal arteries on the right were markedly dilated. The tympanic membranes were unremarkable. The Rinne was negative bilaterally. The neurological examination was negative; the remaining physical findings were consistent with hypertensive heart disease with cardiac enlargement and auricular fibrillation. Otologic consultation at this time recommended x-rays of the sinuses and mastoids and caloric testing. None of these procedures was done. Ophthalmic consultation showed absolute glaucoma O.S. and immature cataracts bilaterally. X-rays of the skull showed extensive Paget's disease involving the vault, particularly on the right side, with extension into the base of the skull. Alkaline phosphatase during this admission was 25.6 units as opposed to a normal of 1.5 to 4 units. The calcium was 9.2 milligrams per cent and the phosphorous 3.0 milligrams per cent. When the diagnosis had been established by x-ray, the patient was discharged from the hospital on phenobarbital 30 milligrams three times a day. On this regime she did well and had no further recorded difficulties with the ears. Subsequent out-patient visits were for unrelated symptoms in the dental and eye clinics. The final admission was in November, 1953, when the patient was admitted with myocardial infarction and died in a short time. Autopsy was performed and the following diagnoses were made:

1. Atherosclerosis, severe, generalized with
 - a) thrombosis of right and left coronary artery
 - b) posterior myocardial infarction.
2. Chronic passive congestion of the liver
3. Pulmonary congestion and edema
4. Paget's disease of the skull
5. Leiomyomata uteri
6. Cholelithiasis and cholecystitis
7. Submucous lipomata of colon.

The neuropathology laboratory made the following report: the skull was found markedly thickened with soft cancellous bone. At the frontal region it measured 3.5 cm in thickness, over the parietal bone, 1 cm. Both internal auditory meati were narrowed and deepened on the internal orifice by circumferential overgrowth of the bone, constricting the VII and VIII nerves as they pass into it. There was a definite internal protrusion of the foramen magnum region into the posterior fossa to the extent of approximately 2 cm. The

neuropathological diagnoses were: 1) Paget's disease of the skull; 2) compression of the VIII nerve bilaterally; 3) basilar impression moderate.

HISTOLOGIC EXAMINATION

The temporal bone was prepared and serial sections were made in the usual manner. Staining of the sections was done with hematoxylin and eosin, and Mallory's stain. In the course of removal of the temporal bone the middle and external ear structures were not preserved so that this examination will concern itself only with the inner ear.

In examining the sections one is impressed by the completeness with which the Paget process has invaded the entire petrous pyramid. Normal bone is not to be seen in any part of the petrous pyramid including most of the cochlear capsule. The process throughout the temporal bone is one of marked activity involving a great deal of osteoclastic activity. Osteoclasts are evident in great numbers and are seen as multinucleated giant cells. There is a moderate amount of vascularity in the entire area. The internal auditory meatus shows mild bony protuberances which allow for compression of the VIII nerve. The facial nerve is uninvolved in its canal. The carotid artery and its canal are also seen to be normal. There has been reconstruction of the entire cancellous portion of the petrous pyramid with Paget bone extending even to the periphery.

In examining the bony capsule, which is the last part of the petrous pyramid to be involved, we find that throughout the entire series of slides the original three layers are no longer distinguishable as such; that is, they have already begun to change and the Paget process with its destruction proceeds from outward in. This in one area is seen to reach throughout the entire bony labyrinth to the membranous labyrinth. The footplate of the stapes has been lost during the preparation; however, the adjacent bone is visible and found to be replaced by Paget bone. There is also involvement of the nerve to the macula of the utricle. This of course would explain the vestibular symptoms which the patient experienced. The membranous labyrinth and cochlea are free of any changes which can be attributed to Paget's disease.

SUMMARY AND CONCLUSIONS

A brief review of Paget's disease, primarily from the otologic standpoint has been presented. Histologic examination of the tem-

poral bone of the patient with Paget's disease who had otologic symptoms is in agreement with the findings of others. No labyrinthine or cochlear changes were found.

52 WEST CENTRAL ST.

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XVII

LARYNGECTOMY: PAST, PRESENT AND FUTURE

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It has been said that in order to see where we are going it is necessary and worthwhile to see where we have been. Therefore, it seems appropriate that on this occasion, when we honor Dr. Schall who has contributed so much to the surgical treatment of carcinoma of the larynx, that we reflect on the past, consider the present, and attempt to look into the future. A reading of the early surgical history is a sobering and comforting exercise and underlines the relative safety and effectiveness of our present methods, crude as they may some day seem to be. The recent gains have been hard won and reflect great credit on all of those who have been associated with them. In obtaining these advances, Dr. Schall has played an important part.

The history of total laryngectomy begins with Patrick Heron Watson¹ of Edinburgh who in 1866 removed a non-functioning syphilitic larynx from a 36 year old man. A tracheotomy was performed one year prior to laryngectomy. After a linear incision was made and the larynx exposed, the trachea was transected and the larynx excised. Watson reported, "The patient rallied from the operation but died some weeks afterwards from pneumonia." This case was unreported until 1881 by Foulis.

Experimental surgery in dogs by Czerny in 1870 proved the feasibility of removal of the larynx and led to the first laryngectomy for cancer, which was performed by Billroth² on December 31, 1873 (Fig. 1). His case was also a 36 year old man who, after having piecemeal peroral removals of "epithelial cancer" and cauterization for some months, was treated by thyrotomy one month prior to laryngectomy. The trachea was anchored with two sutures, and the pharynx partly closed with three sutures. The wound was otherwise

From the Los Angeles Eye and Ear Hospital and the Department of Otolaryngology, School of Medicine, University of Southern California. Presented at a meeting of the Massachusetts Eye and Ear Infirmary Alumni Association, Boston, April 14, 1959.

left open, and a fistulous communication to the pharynx was preserved and later fitted with a speaking tube. The disease was far advanced, the operation conservative, and the patient died of recurrence within seven months. Following this, many other similar operations were performed on the continent and in England, both for carcinoma and sarcoma.

The first extensive operation in which the entire larynx, hyoid bone, part of the tongue, pharynx, and esophagus were removed was by von Langenbeck³ in 1875, using a "T" incision (Fig. 2). Death occurred four months later from lymphatic metastases. Foulis⁴ in 1877, performed a laryngectomy on a 28 year old man after an unsuccessful cricotomy in 1876 and thyrotomy in 1877 for a "papillomatous round cell sarcoma." Death occurred from phthisis in 17 months' time. By 1881 Foulis⁵ was able to collect 32 cases of total laryngectomy, 25 for carcinoma and 7 for sarcoma. Of these, 16 died from the operation and 7 of recurrent disease in a short time. Others died of non-malignant causes or were too recently operated upon to know the result. One, that of Bottini⁶ (Fig. 3) in 1875, for sarcoma in a 24 year old postman was a lasting cure. This patient was known to be alive and well six years later. In 1879, Lange⁷ (Fig. 4) became the first to perform laryngectomy in America. He used a "T" incision and removed the larynx, half the hyoid bone, and the anterior muscles to which the fibrosarcomatous tumor had become attached.

In 1887, the case of the Crown Prince Friedrich of Germany brought the whole subject of cancer of the larynx and its surgical treatment into dramatic world-wide prominence. The net result of this tragedy, among other things, was to tend to discredit the value of biopsy in establishing the diagnosis, for it was long after the onset and after assorted treatments and inconclusive biopsies that the true nature of the disease was established. As Stevenson⁸ has pointed out, Morell Mackenzie was correct in insisting that a positive biopsy should be obtained before a radical operation was carried out. The chief criticism is that he relied too implicitly on a negative report, even though that report was given by Virchow himself.

By modern standards the disease in most of these early cases was far advanced, and the surgical mortality was high. Pneumonia and generalized sepsis were common complications. Many cases had had an unsuccessful preliminary thyrotomy. The wounds were usually left open and were grossly contaminated by infected pharyngeal secretions. From the first, a fistulous communication to the pharynx was planned to accommodate an upward extension of the tracheal



Fig. 1.—Illustration from Billroth's first case of laryngectomy for cancer in 1873.

cannula, facilitating postoperative speech. It is interesting that the thought of rehabilitation was present from the beginning. Gussenbauer designed a tube to transmit air into the pharynx in Billroth's first case.

Early efforts at laryngectomy, therefore, were as discouraging as the first cases of thyrotomy, and progress until 1900 was very slow. Gluck and Soerensen^{9,10} in Berlin perhaps did the most to improve and develop the technical details of laryngectomy. Starting first with and abandoning a two-stage operation, they developed an approach on a wide-field basis with open exposure of the neck. They developed the plan of removing the larynx from above downwards, and of closing the pharyngeal defect before amputation of the larynx. They further developed the idea of suturing the trachea to the skin of the neck. Solis-Cohen¹¹ of Philadelphia, who performed the first successful laryngectomy in America in 1892, modified Gluck's method by suturing the trachea to a cutaneous buttonhole. He thus secured a bridge of skin between the trachea and the pharyngeal wound. Even with these methods, the incidence of surgical mortality and septic complications was high.

It was Mackenty¹² who, by restricting the operation to highly favorable intrinsic cancers and by applying the most careful technical details, demonstrated the true potential of laryngectomy and placed it on a firm basis. The outstanding points in laryngectomy developed by him have been detailed by Lewis.¹³ Antibiotic control of infection and other aids notwithstanding, these are as important and worthy of attention today as they were then. Careful pre-operative medical evaluation was essential, as well as pre-operative attention to dental and oral hygiene. He was an advocate of combined local and general anesthesia and laid great importance on excluding blood from the trachea. To accomplish this, he used with great success the removal of the larynx from below, upwards. He developed a method of abundant drainage of the operative field, the tracheal stay suture, the nasal feeding tube, and the open type of operation with "T" incision. His surgical mortality was low; however, healing by secondary intention was not eliminated, and it has been only since the development of antibiotic control of infection that primary union has become the rule.

In pre-antibiotic days the concern of many was to attempt to minimize delayed healing and its complications by adopting a narrow-field surgical approach. It was hoped that by using conservative skin incisions, by saving extrinsic laryngeal muscles, and by skeletonizing

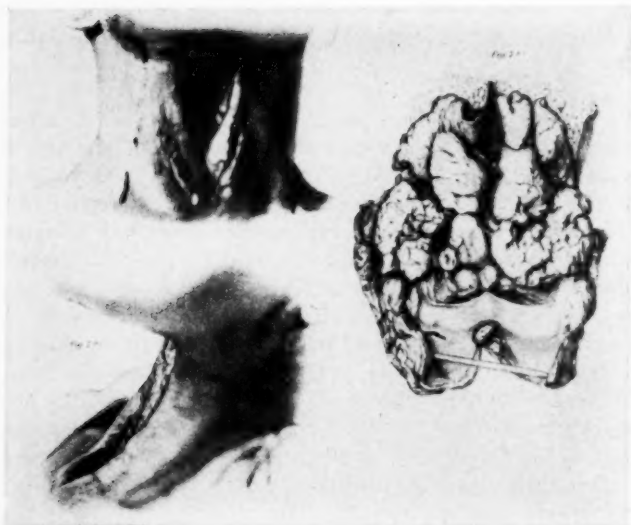


Fig. 2.—Illustration from von Langenbeck's laryngectomy in 1875.

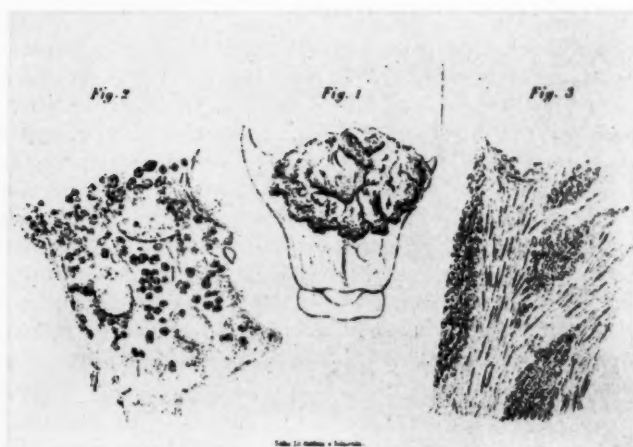


Fig. 3.—Illustration from Bottini's first successful laryngectomy in 1875.



Fig. 4.—Illustration from Lange's first case of laryngectomy in America, 1879.

the larynx, support could be added to the pharyngeal closure. Fascial planes in the neck would be left undisturbed, and healing would be assisted. This technique was advanced on the grounds that only selected intrinsic cases were suitable for laryngectomy, and that if the disease extended beyond the laryngeal cartilages the case was incurable by operation. Today we know this is not true. By wide-field laryngectomy, cases of marginal recurrence have been practically eliminated, and by a further extension of the operation many cases with lymph node metastasis have survived.

Control of infection has made these narrow-field approaches no longer necessary. Wide exposure of the anterior of the neck with either a "T" or a "U" flap will prove superior. In a case suitable for simple laryngectomy (Fig. 5, 6, 7) the strap muscles, hyoid bone, larynx, pre-epiglottic space, and upper tracheal rings are removed in one specimen, the defect closed, the wound drained, and primary healing may be expected in 7 to 10 days (Fig. 11). If there is an indication for a radical neck dissection, the operation on the above plan with slightly modified incisions may be extended to include also the thyroid lobe, sternomastoid muscle, internal jugular vein, all associated lymphatics, and the submaxillary salivary gland (Figs. 8, 9, 10). It is doubtful that all cases requiring laryngectomy need a simultaneous neck dissection, but those with a marked extra-cordal extension or of extra-cordal origin are best treated by a primary

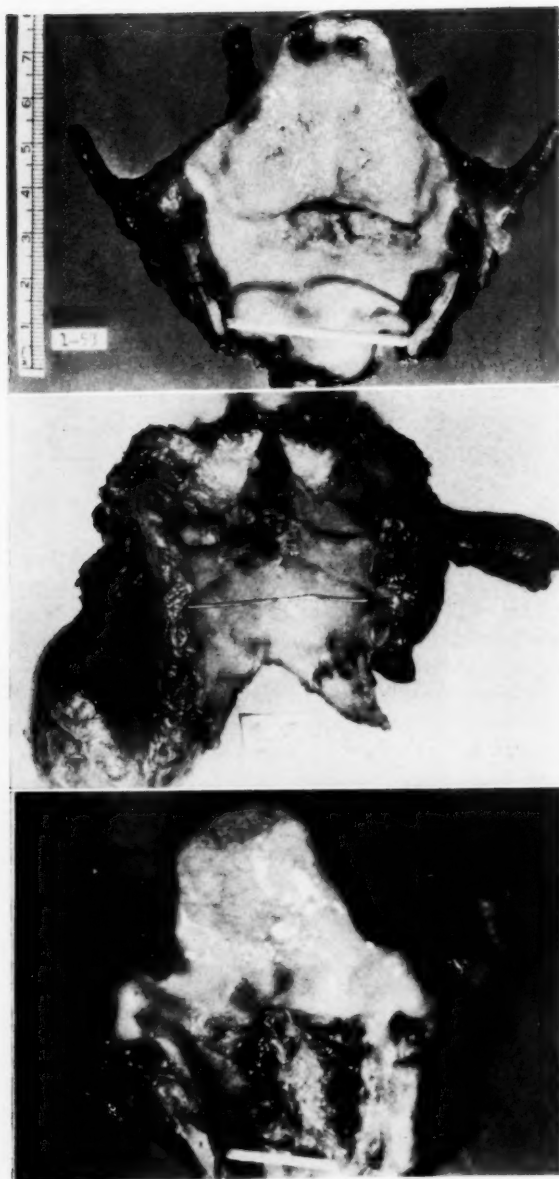


Fig. 5.—Cordal carcinoma involving the major portion of the right true vocal cord, anterior commissure and anterior one-third of the left true cord. There is also an extension into the fundus of both ventricles. Wide-field laryngectomy was performed on May 15, 1953, with no recurrence to date.

Fig. 6.—Primary carcinoma of the interarytenoid space, an uncommon site. There is both proliferation and ulceration, and the process has spread to the posterior portion of the right true cord and ventricular band. The larynx has been opened anteriorly, and there is a large calcified right thyroid lobe attached to the specimen. Wide-field laryngectomy June 25, 1957, with no recurrence to date.

Fig. 7.—Extensive right cordal carcinoma with obliteration of the ventricle, extension to the left and massive subcordal invasion. Wide-field laryngectomy September 9, 1949. The patient developed mediastinal and pulmonary metastases and died July 15, 1950.



Fig. 8.—Extensive right cordal carcinoma with marked subepithelial vestibular invasion obliterating the ventricle. There is also a superficial extension into the left ventricle and cord. The internal opening of tracheostomy is shown. Wide-field laryngectomy and right radical neck dissection was performed on August 25, 1958.

Fig. 9.—Moderately large proliferating carcinoma of the right ventricular band and epiglottic base. Wide-field laryngectomy and right radical neck dissection March 2, 1959. Postoperative picture Figure 12.

Fig. 10.—Extensive carcinoma of the left pyriform sinus previously reported (*Arch. of Otolaryngol.* 64:258-266, 1956). Pharyngolaryngectomy and left radical neck dissection was performed May 5, 1954. The patient remained well but died February 3, 1959. An autopsy revealed adenocarcinoma of the head of the pancreas with pulmonary, hepatic and adrenal metastases. There was no gross or microscopic evidence of recurrence of epidermoid carcinoma of the pyriform sinus.

one-stage operation as advised by Clerf,¹⁴ Ogura,¹⁵ Pietrantonio and Fior,¹⁶ Putney,¹⁷ Schall,¹⁸ Work,¹⁹ and others.

The procedure to be followed becomes more difficult in midline or near-midline growths which threaten either or both sides. It has been shown that lesions decidedly unilateral but approaching the midline may spread to the contralateral lymph nodes without involving those on the side of the primary lesion.²⁰ Perzik²¹ has reported that bilateral jugular vein resection is not considered indicated except for bilateral palpable adenopathy. The surgical risks of laryngectomy and unilateral radical neck dissection are insignificantly greater than those of simple laryngectomy alone, and improved statistics on survival may be expected by this operation in selected cases (Fig. 12). Serious complications, however, may result from simultaneous bilateral jugular vein ligation. These risks might be lessened by conditioning the collateral venous circulation by a preliminary ligation of one jugular vein, several weeks prior to laryngectomy and bilateral neck dissection.

Let us turn to the advances with which Dr. Schall²²⁻²⁸ has been closely associated. He has always advocated that the surgical approach be radical, and that it should be conceived to conform to the pathological realities. Starting in the pre-antibiotic era he advocated the wide exposure of the larynx and, particularly, the need for removing the prelaryngeal strap muscles. This was based on the need for a wide exposure of the disease and its extensions and facilitated the plastic and functional results. With removal of these muscles and careful attention to the tracheal stoma there is rarely a necessity for a postoperative cannula. Removal of the muscles and a portion or all of the hyoid bone permits the removal of the entire pre-epiglottic space which may be a site for early non-metastatic direct extension of anterior glottic or, particularly, supraglottic lesions. He also demonstrated that the muscles may be a site for neoplastic extensions. Since they have no value after the larynx has been removed, it is best to do away with them. This approach has led to the extension of the operation to include the lymphatic vessels and nodes of the neck, and has eliminated marginal recurrences in the region of the tracheal and pharyngeal stomata which were common with narrow-field techniques. Control of infection has added greatly to the safety of this type of an extended operation. A wider operative approach has also made possible the successful treatment of more extensive cordal and extracordal disease, previously thought to be incurable. Sad experience has shown that poor results are to be anticipated in these cases of extensive disease by radiation.



Fig. 11.—Patient on the 6th postoperative day after wide-field simple laryngectomy, using a "U" flap.

Fig. 12.—Patient on the 8th postoperative day after laryngectomy and right radical neck dissection, using a modified "U" flap. Moderate edema of the right side of the face and submaxillary area is present.

These ideas are of a technical nature and, although of great practical importance, are of little interest to the patient. Combined with these efforts, Dr. Schall has since the beginning recognized and emphasized the human aspects of the problem. The work that he has done in promoting speech rehabilitation of those handicapped by loss of the larynx has been a most important and worthwhile contribution, a contribution which makes the surgical efforts more meaningful. Systematized instruction in esophageal speech can restore almost all of these patients to a useful capacity in life. By his efforts he has proved without a doubt that the laryngectomized patient is fortunate and not to be viewed with pity.

Predicting the future of laryngectomy, when our vain hope is to resolve the nature of cancer at a fundamental and etiological level, is to some extent hazardous. However, etiological revelation is one thing and practical application of that knowledge is another, and it would seem that for the predictable future, fundamental advances notwithstanding, the present trends will continue. It is of interest that the earliest operations which are now applied to malignant neoplastic disease were performed for non-malignant conditions,

and it may be that the need for partial and total laryngectomy will outlast their application to carcinoma. For example, thyrotomy may be used for laryngeal reconstructive procedures, and laryngectomy may be required to remove a non-functioning, potentially dangerous non-carcinomatous larynx.

It is clear today that neither radiation nor surgery is the absolute solution to the problem of carcinoma of the larynx, but of the two methods we can in general expect greater survival rates with surgical treatment. There is a tendency to advise radiological treatment for the most favorable intrinsic cases and to use radical surgical extirpation for the more advanced, reversing the trends of the pre-antibiotic era. In ideal intrinsic cases, thyrotomy and cordectomy should continue to be useful, especially in the young because of the potentially high cure rate. Radiation may find its widest application in those cases where the volume of tumor is small and which appear to require an intermediate operation between thyrotomy and laryngectomy.

The continued and expanded use of primary laryngectomy combined with neck dissection, both prophylactic and curative, should improve the survival rate and may add 10 to 20% to the 60 to 65% five-year survival rate of wide-field simple laryngectomy. If the safety of the one-stage laryngectomy and bilateral radical neck dissection can be improved, using this operation in appropriate cases should secure a further increase in the survival rate. Those cases producing distant metastasis or those with subglottic involvement which tend to invade the peritracheal lymphatics and spread to the mediastinal lymph nodes can not be salvaged by any known form of treatment and will continue to take their toll (Fig. 7).

Continued use of esophageal voice training should provide a means by which most subjects can regain useful speech. The diagnostic and pre-operative inquiries in a case requiring laryngectomy have a tendency to select those self-determined individuals who may be expected to make a completely satisfactory speech recovery. For those cases who have difficulty for one reason or another in learning esophageal speech, the tracheo-esophageal fistulizing operation recently described by Conley²⁹ may prove useful. For those candidates who shrink from laryngectomy even though it means greater life expectancy, the example of those who have survived and have made a satisfactory adjustment should provide encouragement at a time when it is needed.

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XVIII

PSYCHOGENIC HEARING LOSS IN CHILDREN

A PRELIMINARY REPORT

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During the last few years, at the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary there has been encountered a rather perplexing problem, that is, children with a distinct, yet non-organic hearing loss.

Functional hearing loss has been recognized for at least 100 years,¹ and a great deal has been written on the subject, mostly in post-war eras. This material, however, deals in the main with veterans, having but few references to children.¹⁻³ Thus, as the first few cases were met in the Foundation, some question was raised as to the correctness of diagnosis, but with subsequent patients and a resulting increase in interest, the entity became more apparent.

The purpose of this paper is to make a preliminary analysis of this problem. In order to do this properly, a definition of terms should be set forth. Webster defines a child as a son or daughter or one who is weak in knowledge, experience, judgment or attainments. Included in our cases are a few children in their late teens or their early twenties, but who have been in school steadily and under constant parental supervision in a home environment. Excluded are any who have had military service. Turning a few more pages in Mr. Webster's book, the word psychogenic is found, and when bisected, psycho means mind, signifying relation to mind or soul, and genesis denotes origination. Thus psychogenic refers to that which originates in the mind. Again, this is a broad definition and here includes the unconscious, that is, conversion hysteria, and the conscious, which is malingering. In either case there is a secondary gain, but it is rarely apparent to the otolaryngologist. However, there are some clues

From the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary.

20 decibels to total deafness which differ from those of Johnson et al. who, in a study of adults, found the average loss to be 35 decibels.⁴

APPROACH TO THE PROBLEM

This hypothesis leaves many questions among which are a few that we feel have been at least partially answered, but only after thorough investigation. This was not a one man job. To attack the problem, as suggested by Martin, a team was needed.⁷ Ours consisted of five members: the otolaryngologist, audiologist, educational psychologist, social service worker, and psychiatrist.

PROCEDURE

Most of these patients were seen initially in the Ear, Nose, and Throat Clinic or the Winthrop Foundation at which time the otolaryngologist was the first one to evaluate them. A few cases came from private practice. Of course the patient's chief complaint was that of hearing loss or deafness which may have been of many years' or short duration. Usually it was at this visit that some clue was afforded the doctor indicating a possible psychogenic deafness. This might be only a hunch derived from some of the clues and hints suggested above. Often the patient brought a school hearing test showing bilateral 80 decibel nerve type of loss, yet sat across the room from the examiner and without any difficulty participated normally in a conversation when the examiner's voice was at normal or subdued intensities. In the younger patients one could easily trick them by talking about some familiar and enjoyable subject.

A careful history including infection, pre-natal influences, RH factor, viral diseases, jaundice, anoxia, etc. was recorded, and then a physical examination of the ear, nose, and throat was done. In general the physical examinations were within normal limits. Cases with significant pathology were excluded.

The next member of the team to come into contact with the patient was the audiologist who used pure tone, air and bone conduction, SRT and Pb tests. In younger patients this was done in two sittings. Responses to the pure tone, as a general rule, were inconsistent, and almost always a note would come back on the audiogram, labelling it unreliable. Although the pure tone tests usually were much lower than the SRT tests, this is not infallible as a true conversion hysteric can easily hurdle this barrier.⁵ As seen by Johnson and his coworkers in adults, we found that the occasional patient did not

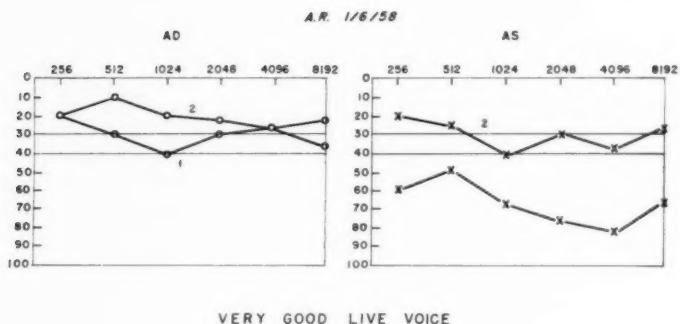


Fig. 2.—Audiograms showing discrepancy between pure tones on same date.

answer any of the words correctly on the SRT, or that he answered only one syllable of every word, and when pressed to guess at the other half, made up a ridiculous sound. Tuning fork tests were practically valueless except that their bizarre results made one suspicious of functional hearing loss.

At this point cases not showing normal hearing were also tested with the psychogalvanic skin resistance method. In 1947 Michaels felt that the PGSR, properly performed, was conclusive and particularly suitable in evaluating claims of total unilateral or bilateral deafness.⁸ Knapp, in 1950, found the PGSR not infallible, but of sufficient value to discover normal hearing in adults. When these cases were followed by psychotherapy, 90% or more gained normal hearing.⁹ (It is to be noted that the PGSR must be given by a person trained in this type of work.)

After the otolaryngologist and audiologist had discussed the case and no organic basis for a hearing loss was evident or the audiometry disclosed normal hearing in one or more of the tests, it was felt that psychogenic hearing and mental retardation or both must be ruled definitely in or out.

The third and fourth members of the team, the educational psychologist and the social worker, then came into play. Since retarded and borderline mentally defective patients give audiometric responses similar to those of hysterics,⁴ it was necessary for the psy-

chologist to ascertain the child's level of intelligence. Moreover, carefully selected psychometric instruments sometimes gave insight into personality dynamics involved in the psychogenesis of the hearing loss. We found it to be helpful also in children of superior intelligence who were doing poorly in school. As an example, we had school referred cases of students failing reading who were sent to ascertain the presence or absence of a hearing loss. If a child has normal hearing, the dynamics of the situation may be such that his reading ability is superior to that of the rest of the class, but when the teacher quizzes him about one page of the reader, he has forgotten it because he is reading far ahead of his classmates.

The social worker assumed much of the responsibility in searching for causative factors in the domestic environment and in helping families to accept appropriate aid from other agencies, as recommended by the team. She accomplished these objectives through interviews with the parent, child, and often with school personnel and community agencies familiar with the family. The social worker aided the family in understanding the patient's problem and psychologically supported their efforts to carry out team recommendations through recognition of their feelings and through guidance. Sometimes simply talking to the social worker appeared to be therapeutic for the parents. The social worker's goal always was to help parents meet their child's total needs as far as was possible without damaging the family itself.

When the diagnosis of psychogenic deafness was certain, an attempt at therapy was made. By "treating" the earlier cases with repeated pneumatic otoscope examinations and assurance from the otolaryngologist that they would get well and repeated audiometry, many showed an improvement to normal hearing levels. This method of assurance was also used by Rosenberger and Moore in conjunction with narcosis.¹⁰ In other words, we were giving these children a chance to "back out gracefully" as mentioned by Johnson and his collaborators, who felt that this type of case represented malingering. They thought that hystericals would give up their hearing loss through psychotherapy if opportunity and motivation were provided.⁴ But the team as a whole was not happy with the situation and felt that backing out gracefully (even if malingering) solved only the present problem; the deeper, more basic stimulus was still burning, and complete treatment should include eradication of the underlying cause.

Few of our patients have had psychotherapy, and those few have not yet completed treatment. The majority of patients seen in the

last two years have had one or two diagnostic interviews with the psychiatrist. In all these cases psychiatric disturbances were noted, and when indicated and providing the parents were willing, referrals for psychotherapy were made with either the OPD or private physicians.

REPORT OF A CASE

A.R., a 17 year old young lady, was first seen in October of 1957, complaining of hearing loss since the age of 13 and bleeding from her left ear. She used wool in the left ear to stop the bleeding, but it was so bad that it ran down the side of her face. At the age of three she scratched her right ear drum with a bobby pin, but suffered no ill effects. Menarche occurred at the age of 13, and it was at this time that she first met her father. At this time also she had abscessed ears treated successfully with drops. Three months ago she failed a school hearing test. The family history disclosed that both parents have asthma; the mother has polycystic kidneys. There is no history of deafness. The physical examination was normal. She was told to return immediately when bleeding recurred, but with every return, the bleeding had stopped. Psychiatric study indicated many features suggesting psychoneurosis: semidelinquent behavior, multiple phobias, a tendency to somatize, and a great difficulty in controlling her aggression impulse.

SUMMARY AND CONCLUSIONS

Psychogenic deafness is an entity afflicting children as well as adults. Malingering and conversion hysteria both are included in this disturbance. "Backing out gracefully" toward normal hearing is successful, but treats the symptom while the basic underlying psychological cause is left smouldering. It must be discovered and treated. To meet this end the co-operative efforts of the otolaryngologist, audiologist, psychologist, social service worker, and psychiatrist are moulded into team work. A good team needs a strong bench, and this should include the family doctor, welfare agencies, school systems, and the family itself. A typical case record is presented.

243 CHARLES ST.

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XIX

RESULTS OF NINE HUNDRED AND THIRTY-NINE STAPES MOBILIZATION OPERATIONS

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Stapes mobilization is a procedure subject to modification from case to case because of the many variables imposed by individual differences in ossicular structure.¹ Anatomically the topographies of stapedial regions are nearly as variable as thumb prints from patient to patient. Visualization of the stapes may be easy, difficult or impossible. The extent of otosclerotic formation varies within wide limits. Preoperatively the area of fixation is quite unpredictable. Consequently, so is the prospect of achieving satisfactory hearing improvement. These and other factors have resulted in the necessity for surgeons to vary their techniques, approaches, and prognoses with respect to the stapes mobilization operation.²

Despite these variables which have prevented a standardization of the procedure, it would seem that a review of a large number of operations performed within a relatively short period might be fruitful. Therefore the purpose of this paper is to summarize certain aspects of the stapes mobilization operations performed thus far in the Department of Otolaryngology at the State University of Iowa. From October 1, 1954 to February 28, 1959, at which time preparation of the following summary was begun, 939 operations had been performed.³ It was hoped that from a review of these operations some trends might emerge which would reveal significant relationships between various factors associated with the operation and results derived therefrom.

DATA COLLECTION AND PROCESSING

Preparation of the data has been facilitated through the use of IBM cards. Patient identification, preoperative case history, surgical and audiological information have been encoded by one of us (Iles)

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in the Department of Otolaryngology. One hundred fourteen of the 160 available columns on two IBM cards are utilized for this purpose at the present time. For each postoperative medical and audiological re-evaluation of the patient a similar pair of IBM cards has been punched. As of February 28, 1959, each operation has been followed by an average of four audiological evaluations. Thus an average of ten cards have been encoded for each operation. The nearly 10,000 IBM cards were then sorted, matched, counted and certain factors recorded thereon were correlated by the University Statistical Service. The Director of our Audiology Section (Shapley) assisted in the final organization of the findings and the preparation of this report.

DATA DEFICIENCIES

Care must be exercised concerning the interpretation of certain numerical values obtained from a survey of this type. A single mean does not reveal anything about the systematic changes in values from which the mean was computed. Within a period of nearly five years, systematic changes have occurred that deserve consideration. Necessary modifications in a particular mobilization operation, for example, are difficult to encode but they may materially affect operational success. An increase in the skill of the surgeon cannot easily be expressed as a number, but greater skill undoubtedly improves the surgeon's rate of success. The same may be said with regard to improved instruments for visualization of the operative field and for mobilization of the stapes. As experience is gained by the surgeon, he formulates impressions that are difficult to quantify, but which assist him to become more selective with respect to the patients he is willing to accept for the procedure. His experience also leads him to obtain from his more recent patients additional case history, surgical, and audiological information—information that was not and cannot now be obtained for previous operations.

Some of these systematic changes which occur with the passage of time are not readily quantified and are not represented by punches in our IBM cards. In a sense, therefore, they represent deficiencies in terms of the arithmetic treatment of our data. However, in the discussion to follow, attention is directed, wherever it seems advisable, to the influence of such variables upon the computed values included in the survey.

DATA REDUCTION

Although the data currently encoded on our IBM cards can provide useful answers to literally hundreds of specific questions relating

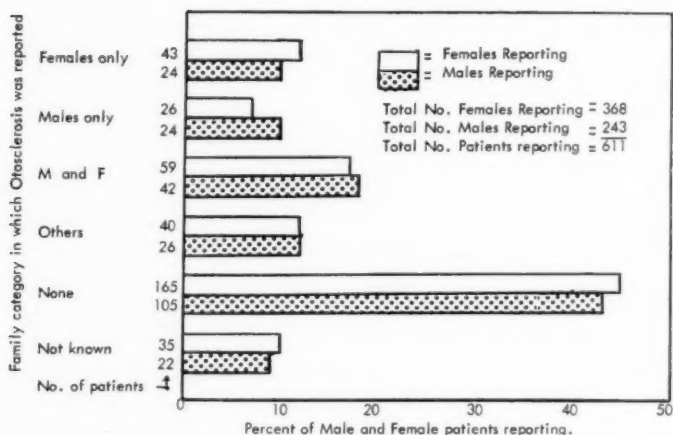


Fig. 1.—Incidence of otosclerosis in the immediate families of 611 patients who had stapes mobilization operations.

to our mobilization procedures, our first major analysis has been restricted to certain issues which seem to us to be of particular relevance at this time. The data has been investigated with respect to four general areas: 1) case history information; 2) surgical success in terms of pure tone audiometric results; 3) maintenance of success; and 4) certain audiological relationships. Findings in each of these areas and our interpretations of them are presented below.

I. CASE HISTORY INFORMATION

a. *Sex Ratio.* The 939 operations upon which this report is based were performed on 611 patients. Of these 60% were females, hence there is a ratio of three to two in favor of females. Goodhill and Holcomb⁴ have reported a similar ratio for operated patients. Although this ratio departs substantially from the incidence ratio which is usually reported^{5,6} it should be pointed out that the sex ratio for operated patients does not necessarily represent the sex ratio for incidence.

It is of interest to compare this 3:2 ratio of females to males for stapes mobilization patients, to the 4:1 ratio for our fenestration patients.⁷ The lower ratio for the former cases probably reflects our

willingness to attempt mobilization even where there is a certain amount of bone conduction loss. With our selective criteria thus relaxed, more men with slight to moderate occupational losses have been accepted as candidates for mobilization.

b. *Heredity.* An attempt to establish from our data some hereditary trends met with disappointment. The distribution of otosclerosis within the immediate families of the patients included in this study is shown in Figure 1. Two results of interest are: 1) from information provided by the limited number of patients definitely reporting the existence of the disease in their families, no obvious tendency is shown for otosclerosis to be predominant in either sex; 2) nearly half of the patients declared there was no otosclerosis in their families. This second result may be viewed with suspicion, but it is consistent with a previous survey concerning the fenestration operation.⁸ It must be remembered that a patient's report about the otosclerotic condition of members of his immediate family is subject to all the usual uncertainties and errors inherent in recall and ignorance of the facts. It seems probable the percentage of patients reporting no otosclerosis in their families would be reduced if they all were actually aware of the facts.

c. *Tinnitus.* Currently we are trying to establish the incidence and nature of pre- and postoperative tinnitus in stapes mobilization patients. Six categories of tinnitus ranging from "low pitched and pulsating" to "high pitched and constant" have been made available to the patient. A complete breakdown of the pre- and postoperative tinnitus information for the 939 operations is shown in Figure 2.

Tinnitus of any sort was denied prior to 224 operations. This represents 24% of the total number performed. Various types of tinnitus were reported prior to 497 operations (53%). Prior to the remaining 218 operations (23%), the existence of tinnitus was not determined.

Postoperatively the existence of tinnitus was denied in 71, or 8% of the cases. (Of these, 54 had denied its existence preoperatively.) Thirty-seven (3.4%) reported tinnitus of some type postoperatively, but of the remaining 831 (88.6%) operations, the existence of tinnitus was not determined.

From these results little can be said regarding the probable effect of mobilization on tinnitus. The most important conclusion from this analysis is that we need in our records more consistently recorded information about tinnitus.

Patients who had this type tinnitus PREOPERATIVELY	Had this type Postoperat- ively									
	None	Low & Puls.	Low & Const.	Low & Occas.	High & Const.	High & Occas.	Yes (Un descr.)	Not Determined	Totals Preop.	%age Preop.
None	54				1			169	224	24.0
Low & Pulsating	2	1					3	19	25	3.0
Low & Constant	2						4	45	51	5.0
Low & Occasional	3			3			1	61	68	7.0
High & Constant	4				3		2	94	103	11.0
High & Occasional	4	1	1		2	1	4	82	95	10.0
Yes (Undescribed)	2		1				8	144	155	17.0
Not Determined							1	217	218	23.0
Totals Postoperatively	71	2	2	3	6	1	23	831	939	
%age Postoperatively	8.0	0.2	0.2	0.3	0.6	0.1	2.0	88.6		100.0

Fig. 2.—Distribution of the types of preoperative and postoperative tinnitus.

II. SURGICAL SUCCESS IN TERMS OF PURE TONE AUDIOMETRIC RESULTS

In a report by Kos to the Triological meeting in San Francisco in 1958, it was pointed out that criteria by which operations are judged successful vary among surgeons to some extent.⁹ As yet there is no universally accepted set of audiometric results by which a given operation is declared a success or failure. However, yardsticks now in general use are gradually becoming less variable, and it seems reasonable to believe that success rates computed by most otologists are at least roughly comparable.

The pure tone audiometric basis upon which our operations are judged a success is the average loss in decibels for 500, 1,000, and 2,000 cycles per second. Three major criteria of success are specified as follows:

Criterion 1. Closure of the air/bone gap—defined as a difference of 10 db or less between the preoperative bone conduction threshold and the postoperative air conduction threshold. Success by this criterion can be obtained regardless of the absolute magnitude of the preoperative or postoperative average pure tone loss by air.

Criterion 2. A postoperative improvement in the average pure tone threshold by air to 30 db or less relative to the audiometric zero reference.

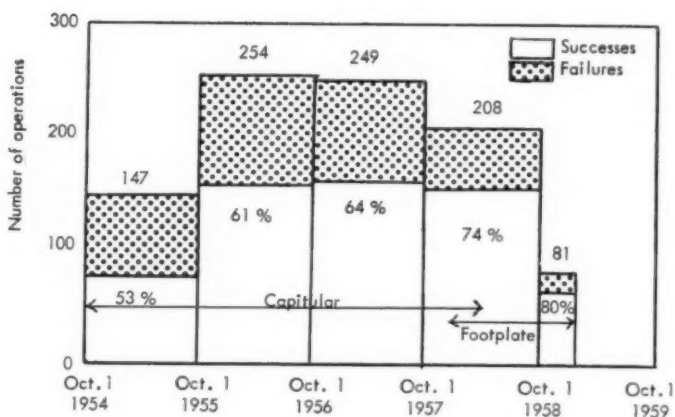


Fig. 3.—Summary of stapes mobilization operations indicated by years since the operation was first performed.

Criterion 3. A postoperative improvement in the average pure tone threshold by air of at least 15 db regardless of the obtained air/bone gap closure or final average air loss relative to the audiometric zero reference.

a. **Overall Success Rate.** Pooling all successful operations, regardless of when performed, by which of the three criteria success was declared, whether they were immediate or delayed postoperative successful level, we have had 612 successful results. This represents 65% of the grand total of 939 operations. As mentioned earlier, however, a lumped value of this sort is misleading for several reasons. By itself, it reveals nothing about changes in the success rate with the passage of time, or changes in instruments, etc. We have, therefore, made use of our IBM facilities to examine our results from different points of view. Figure 3, for example, shows that the success rate, using average pure tone data, has increased from 53% during the first year (1954-1955) the procedure was used, to 80% for the operations thus far performed in 1958-1959. Some of the factors mentioned above have probably influenced this improvement in the success rate. Also shown in Figure 3 is the fact that the capitular technique was used primarily for the first 3½ years, but during the past year the footplate technique has been used.¹⁰

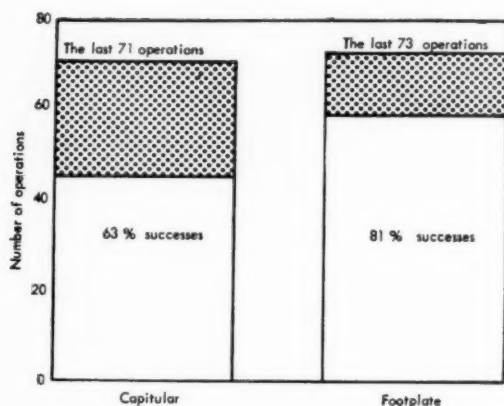


Fig. 4.—Comparison of the rate of successes of most recent operations for capitular and footplate procedures.

The capitular technique employs a pick with which the capitulum of the stapes is engaged through the articular capsule as described previously.³

The footplate technique referred to in this study is comprised of the application of micro-chisels of various angles to the periphery of the stapes footplate or to the junctions of normal and otosclerotic areas on the footplate.² This group also includes a number of anterior-crurotomies not specifically evaluated in this study. A more complete evaluation of the results of these surgical approaches will be discussed below.

Another variable during this period is the improvement in surgical instruments which have been employed since we began using the procedure. For example, a binocular microscope was first put into consistent use about two years ago. Prior to this magnifying loops were used alternately with the microscope during the operation. It seems likely that further increases in success rate also may result from more refined methods of selection based on case history and preoperative audiological analysis. Furthermore, we are mindful of the possibility that other techniques of mobilization such as the use of prosthetic devices may provide an additional increase in the success rate. These possibilities are being explored.

TABLE I
REGRESSIONS WITHIN DELAYED AND
IMMEDIATELY SUCCESSFUL GROUPS

	SUCCESS GROUP		TOTAL
	IMMEDIATE	DELAYED	
Number of Successes	527	85	612
Percentage of Total Successes	86%	14%	100%
Number of Regressions	179	20	199
Percentage of Regressions within Group	34.4%	24%	32.5%

b. *Capitular vs. Footplate Technique.* A re-examination of our successes in terms of the procedure used, disregarding all other factors, indicated that the rate of success for the last 71 capitular operations was 63% as compared to 81% for the last 73 footplate operations. A comparison of these results is shown in Figure 4. On the assumption that optimum surgical skill had been reached for each of these techniques prior to the operations tabulated, and that the persons in each group constituted a random sample of otosclerotic patients, it would seem reasonable to say the footplate technique has definitely provided a greater success rate than has the capitular technique. This finding is consistent with reports of others.^{11,12}

c. *Delayed Successes.* Of growing interest in our clinic is the fact that the ultimate improvement in hearing following stapes mobilization is often not obtained until a few days, or even a month following the operation. As shown in Table I, of the 612 operations that qualified as "successes" at one time or another, 85, or 14%, were not successes at the time of the first postoperative audiometric evaluation, usually administered one to twenty-six hours after the operation. This substantial rate of delayed success led us to discontinue operating room audiometry toward the end of 1958. Complete reliance is now placed on the visualization of stapes mobility through the otosurgical microscope. The occurrence of these delayed successes also seems to be a compelling argument against immediately proceeding with a fenestration operation under any circumstances.

Various factors may be responsible for improvement in hearing over a period of a few days or weeks following the operation. Blood clots in the middle ear, for example, may temporarily prevent freedom of ossicular and round window movement actually made possible by

the mobilization. The formation of an occlusive clot in the external auditory canal may delay hearing improvement. A temporary traumatic loss from the relative violence incurred by the cochlea during the mobilizing process might also delay the ultimate degree of success attained.

It is of interest to note, for example, that during the first year the footplate technique was used, there was a substantial increase in the number of delayed successes. This seems plausible in view of the fact that the footplate technique probably is somewhat more traumatic to tissue and to the cochlea than is the capitular technique.

We know, of course, that the opposite effect also occurs, that is, a successful result may regress to a non-successful level. As shown in Table I, the percentage of regressions (24.4%) that has thus far occurred among the delayed successes is smaller than the percentage of regressions (34.4%) that has occurred in the group that was successful immediately postoperatively. These regression rates of the 85 delayed successes in terms of the capitular and footplate procedures are compared in Table II.¹³

TABLE II
REGRESSION OF DELAYED SUCCESSES

	NUMBER OF DELAYED SUCCESSES	NUMBER THAT REGRESSED	PERCENTAGE THAT REGRESSED
Capitular Procedure	55	19	34.5%
Footplate Procedure	30	1	3.3%
Totals	85	20	24.4%

Of the 55 delayed successes that occurred during the four years that the capitular procedure was used, 19 (34.5%) eventually regressed to a non-successful level. In contrast, of the 30 delayed successes that occurred during the two years that the footplate procedure has been used, only one (3.3%) has regressed. Although those operations using the capitular method have had a longer period of time in which to regress than have those of the footplate method, the fact that the current regression ratio of the footplate method is only one tenth that of the capitular method seems worth noting. At the present time it seems probable that delayed successes following the footplate operation are likely to remain successful longer than delayed successes following the former technique.

d. *Success and Type of Fixation.* Various schemes have been suggested for classifying the nature and extent of the otosclerotic condition in a given ear.^{12,14} It is to be recognized that such classifications are rather arbitrary, but it is also realized that a more objective approach is extremely difficult. A judgment of the nature and extent of the otosclerotic growth at the time of mobilization is subject to wide variations in visualization and impressions from the "feel" of the process through the stem of an instrument. In our cases we did not at first attempt to systematically record the type of fixation observed. Later it became evident that an effort to relate this factor with postoperative hearing improvement might be worthwhile. At the present time we try to list a given otosclerotic in one of eight categories. In only 245 of the 939 operations have we classified the type of fixation. The type of fixation, the number so classified, and the percentage of successful results based on the total that were so classified are shown in Table III.

TABLE III
FIXATION TYPES AND ASSOCIATED SUCCESS RATES

FIXATION TYPE	TOTAL NUMBER	NUMBER OF	
		SUCCESSSES	PERCENTAGE SUCCESSFUL
Anterior	72	47	65%
Posterior	15	8	53%
Anterior-Posterior	84	52	62%
Marginal	19	13	68%
Entire Footplate	22	6	27%
Anterior and Marginal	11	9	82%
Ant. Post. Marginal	22	17	77%

From this table it can be seen that, of the fixation types recorded, where the entire footplate is involved the success rate has been minimal, whereas the greatest rate of success has been with the anterior-marginal cases. For both types, however, the number of operations involved is so small that generalizations at this point are risky.

III. MAINTENANCE OF A SUCCESSFUL LEVEL

Successful results from stapes mobilization need to be evaluated in terms of the period of time they are maintained, as well as in terms

TABLE IV

STATUS OF 612 "SUCCESSFUL" STAPES MOBILIZATION OPERATIONS

PERIOD DURING WHICH OPERATION WAS DONE (OCT. 1 - SEPT. 30)	TOTAL SUCCESSFUL OPERATIONS	KNOWN TO HAVE REGRESSED	SUCCESS MAINTAINED AS OF LATEST VISIT TO HOSPITAL
1954 - 1955	78	39 (50%)	39 (50%)
1955 - 1956	156	65 (42%)	91 (58%)
1956 - 1957	161	52 (32%)	109 (68%)
1957 - 1958	151	35 (23%)	116 (77%)
1958 - 1959	66	8 (12%)	58 (88%)

of immediate postoperative hearing improvement. A patient's enjoyment from surgical rehabilitation of his hearing can soon vanish if the gain is short lived. We are all, therefore, especially concerned about the lasting quality of stapes mobilization.

The present status of the 612 operations that were successful, whether they were immediate or delayed, is shown in Table IV. As of February 28, 1959, 413 are still successful according to the latest information available from each patient. It should be pointed out that although Table 4 indicates that the percentage of maintained successes has increased each succeeding year, eventual regressions within each year-group may ultimately reduce all "maintained success" rates to similar values. More time must pass before a meaningful comparison of the lasting qualities of recent and earlier operations can be made.

IV. AUDIOLOGICAL RELATIONSHIPS

Assessment of the hearing function in terms of thresholds for pure tones will always be of use to the otologist. There is no question about the value of pure tone audiometric data for diagnosis and the determination of surgical suitability. From the patient's point of view, however, an improvement in his hearing for a sinusoidal waveform is, in itself, of little importance. The sounds the patient most wants to hear, and understand, are those of speech.

a. *Correlation Between Average Pure Tone Loss and Speech Reception Threshold (SRT).* We know there is a positive relationship between the average pure tone loss in db for 500, 1,000, 2,000 cps and

the SRT relative to normal hearing. In order to examine the strength of this relationship for otosclerotic patients we have administered SRT tests in 94% of all preoperative audiometric examinations. The degree of success with which the SRT can be predicted from the average pure tone threshold is indicated by the coefficient of correlation between the two. Based on 883 pairs of values, a correlation of .90 was obtained.

That this relationship still holds postoperatively is indicated by a correlation of .92 based on 703 pairs of postoperative average pure tone and SRT values. Thus, from the standpoint of thresholds, in our clinic the average pure tone loss for the three frequencies listed above predicts quite well the threshold loss for speech.

b. *Postoperative Gain in the Speech Reception Threshold.* As indicated above, our criteria for determining the success of an operation rest upon the improvement in the average pure tone thresholds. Since the preoperative and postoperative SRT values correlate highly with their respective average pure tone thresholds, the difference between these SRT values (hereafter referred to as the SRT gain) should also provide a measure of the audiological success of a stapes mobilization operation.

For various reasons we have not been able to obtain the SRT gains for every operation. Nevertheless, we have recorded the SRT gains, some of which were negative, resulting from 687 operations. Of these 687, 33% resulted in SRT gains of 11 to 20 db. Another 28% obtained gains of 21 to 30 db. The maximum gain for any patient was 55 db, the maximum loss was 25 db.

c. *Correlation Between Air/Bone Gap Closures and SRT Gains.* In view of the high correlations preoperatively and postoperatively between average pure tone thresholds and their respective SRT values, we wondered if a close relationship also existed between SRT gains and air/bone gap closures. The overall correlation based on 684 cases was .87 which suggests the air/bone gap closure is a rather good indicator of the SRT gain to be expected. In this study we also looked at the correlations associated respectively with the capitular and footplate techniques. It was interesting to note that with 475 capitular cases, a correlation of .86 was obtained between air/bone gap closures and SRT gains, whereas a correlation of .92 was obtained between these two factors for 187 footplate procedures. It would appear then that SRT gains can be more confidently predicted from the air/bone gap closures obtained in footplate operations than from capitular operations.

TABLE V
DISCRIMINATION GAINS AND LOSSES ON SUCCESSFUL OPERATIONS
ONLY ACCORDING TO OUR MOST RECENT TESTS

NET GAIN OR LOSS IN DISCRIMINATION SCORE IN %	CAPITULAR PROCEDURE		FOOTPLATE PROCEDURE	
	NO.	%	NO.	%
-40 to -31	2	0.7	0	0
-30 to -21	2	0.7	2	1.6
-20 to -11	6	2.1	10	7.8
-10 to -1	60	20.8	35	27.3
0 to 10	186	64.2	72	56.2
11 to 20	25	8.6	5	3.9
21 to 30	6	2.1	2	1.6
31 to 40	1	0.3	2	1.6
41 to 50	1	0.3	0	0
Total	289	100.0	128	100.0

d. *Effect of the Operation on Discrimination.* Although a measurement of hearing improvement using threshold gains, either for pure tones or for speech, is useful for evaluating the success of an operation, these threshold improvements do not tell the whole story. Perhaps it should be repeated that as far as the patient is concerned, a gain in his average pure tone threshold is of no value to him unless he maintains his ability to understand speech.

The best test of discrimination (i.e. understanding speech) in use at the present time utilizes a phonetically balanced list of words from which a discrimination score is obtained. This test provides a measure of a patient's ability to understand speech at an intensity level well above his SRT. The otosclerotic patient without cochlear or neural involvement may be expected to have a relatively high preoperative discrimination score. For this reason a postoperative improvement in discrimination is not probable and is seldom expected. A postoperative discrimination score equal to the preoperative score permits a gain in the SRT to be fully effective, but a significant loss in discrimination may detract from an SRT gain. In order to assess the overall effect of stapes mobilization on discrimination, we examined the discrimination gains and losses for the 591 cases for which we had this information. Eighty-six per cent obtained immediate postopera-

tive discrimination scores that were within plus or minus 10% of their preoperative scores. (Although a 10% loss or gain in discrimination is usually regarded as a significant change, a more detailed breakdown in these scores has not yet been made.) It was also found that 70% obtained and maintained postoperative scores equal to or better than their preoperative scores.

According to the most recent tests, of the 289 originally successful capitular procedures, 246 patients (85%) still have discrimination scores within plus or minus 10% of their preoperative scores, and 33 (11%) have discrimination gains of 11% or more. These results are shown in Table V. Similarly, the most recent tests on patients who had the 128 originally successful footplate operations have shown that 107 (84%) still have discrimination scores within plus or minus 10% of their preoperative scores, and 9 (7%) have discrimination gains of 11% or more. From these results it would seem that neither operative technique will substantially deteriorate discrimination scores for the majority of cases.

SUMMARY

An assessment of nearly five years' experience with the stapes mobilization procedure, during which 939 operations were performed, has provided us with certain facts and impressions that have been discussed in this report. In summary, these are:

1. For the purpose of tabulating and summarizing medical and audiological information from a large number of operations, a card punch system (such as IBM) is indispensable for stapes mobilization records.
2. A greater awareness of the many variables involved in the procedure has resulted from this study.
3. Of 611 patients, 60% have been females.
4. Insufficient data was available to ascertain a positive hereditary link.
5. The significance of tinnitus preoperatively and postoperatively has not been satisfactorily ascertained.
6. Although overall success rates and similar pooled information is of limited value, a success rate of 65% can be reported.
7. It appears from these data that the footplate technique has significant advantages over the capitular technique.

8. Because 14% of all successes were delayed, the usefulness of surgical audiometry is limited. The occurrence of these delayed successes constitutes an argument against immediately proceeding with a fenestration procedure in the event of an apparent failure of stapes mobilization. Visualization through the otosurgical microscope is more reliable.

9. In general our records indicate successes in mobilization are directly related to the extent of the otosclerotic process.

10. Of the 612 successful operations, 413 or 67% remain successful, as of February 28, 1959, according to our criteria.

11. The correlation between average pure tone losses and speech reception thresholds is high, as is the correlation between air/bone gap closures and SRT gains.

12. Sixty-one per cent of the 687 operations on which we have SRT information have had a gain of 11 to 30 db. This compares well with the 65% overall success rate computed on the basis of the average pure tone gains.

13. A correlation between air/bone gap closures and SRT gains is consistent with correlations between similar measures of hearing improvement.

14. The stapes mobilization procedure has not substantially affected the speech discrimination scores following the majority of operations.

These are a few of the aspects of stapes mobilization procedures about which we have some information. A more thorough analysis obviously would be desirable and perhaps much more helpful in steering a course more directly to better selection criteria and to greater surgical success. The quality and quantity of results appear to be acceptable, but regressions continue to subtract appreciably from the durability of successes.

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SOME UNSOLVED PROBLEMS OF
STAPES MOBILIZATION

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It seems appropriate at this time of tribute to our chief, Dr. LeRoy A. Schall, to convey to you some of the rather complex problems of stapes mobilization as observed from our experience at the Massachusetts Eye and Ear Infirmary. This procedure was introduced here in 1955 by Dr. Philip E. Meltzer after careful consideration of Rosen's¹ original report of purposeful mobilization of the stapes for deafness due to otosclerosis. Although the purpose of this paper is not primarily a statistical report I have drawn on an analysis of 626 cases from both the Winthrop Foundation and personal files in order to point out some of the difficulties encountered in the procedure of stapes mobilization. The surgery was personally done in approximately 310 ears, the remainder having been performed by one of the other members of the Winthrop Foundation team.* It is hoped that a study of this series may serve to delineate some of the problems of mobilization surgery and shed some light on its usefulness and limitations as a result of the techniques used to date.

These results represent all cases done from the beginning by a group of five otologists during the period when each was developing his familiarity with the techniques of stapes mobilization. All types of otosclerotic ears are included, many of which were severely deafened and could not be expected to reach the practical level of hearing. In this sense, then, many of these operations were purely exploratory in nature.

The techniques of mobilization consisted of the original method of Rosen by indirect pressure at the stapedial neck, needling of the stapedial capitulum, and the application of pulsating transincudal force. Direct footplate manipulations, utilized during the latter part of the series, consisted of the use of probes, excavators, needles for trephining the footplate, and, in isolated instances, the variously

* Dr. Philip E. Meltzer, Dr. Francis L. Weille, Dr. Bernard Zonderman, and Dr. Robert E. Klotz.

shaped Herrmann chisels. The chisel technique has been used sparingly since it became evident, early in the course of its use, that an otosclerotic lesion so thick and widespread as to require this type of attack would not only increase the trauma to the stapediovestibular joint but would be unfavorable for mobilization because of the likelihood of early closure. The majority of footplate work entailed either the direct application of force to the area of fixation with a blunt mobilizer, or weakening of the otosclerotic focus at the footplate margin by careful removal of bone with a sharp probe.

CRITERIA FOR EVALUATION AND REPORTING OF RESULTS

For purposes of this report I have tabulated the operative results of all classes of otosclerotic ears without regard for suitability according to Shambaugh's classification. In most instances the audiograms were correlated with both speech discrimination and speech reception threshold tests. The operated ear was classified as improved, a failure, or made worse. Those patients who were improved were subdivided into Group A, consisting of those ears that gained an average of at least 10 decibels for the frequencies of 500, 1000, and 2000 cycles per second and in addition reached or surpassed the 30-decibel-level average for these same frequencies. In group B are those patients who failed to reach the practical 30-decibel level but gained an average of more than 15 decibels in the frequencies of 500, 1000, and 2000 cycles per second.

Figure 1 shows a pre-operative and postoperative audiogram of one case classified as improved in Group B. Those patients whose gain was insufficient to fall into the improved group, or whose loss as a result of surgery was less than 10 decibels for the three speech frequencies, were called failures. An ear that showed a persistent postoperative loss as a direct result of the operative procedure of an average of 10 decibels or more for the frequencies of 500, 1000, and 2000 cycles per second was classified as having been made worse.

The audiogram in Figure 2 exemplifies the fate of an ear made worse. This is the most severe loss in this series and is the only dead ear out of the 626 cases.

PREVIOUSLY REPORTED RESULTS

During the evolution of various mobilization techniques there have been fluctuations in the percentage of good results, but the overall average has tended to increase with the experience of the surgeon

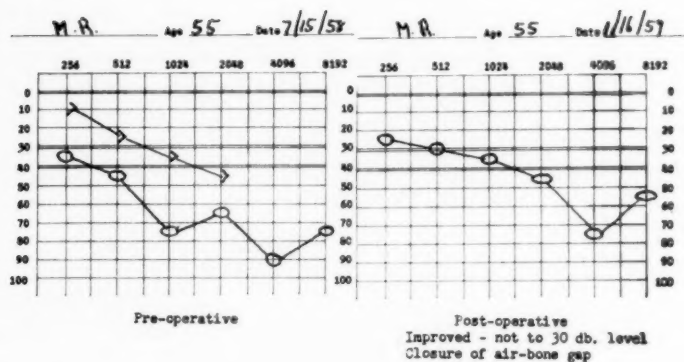


Figure 1

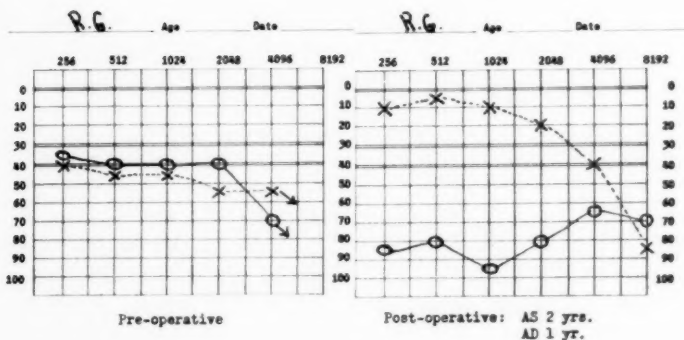


Figure 2

and the use of the operating microscope that allowed direct visualization and exploitation of the footplate.

Rosen² reported that 77 per cent of 254 patients operated on one or more times had improvement in hearing; Bellucci³ had 55 per cent good results with direct footplate mobilization as compared to 29 per cent with indirect methods; Meurman and Meurman⁴ had 60 per cent good initial results in their third year of performing this surgery; Schuknecht et al⁵ had 72 per cent good initial results with the use of the chisel technique in 73 cases; Kos⁶ on the basis of a

TABLE I
INITIAL RESULTS IN 626 EARS

	NUMBER OF CASES	PERCENTAGE
I. Improved		
A. to 30 db level	304	48.56)
B. 15 db gain	51	8.14)
II. Failures	259	41.37)
III. Made worse	12	1.91)
		56.60
		43.28

TABLE II
IMPROVED GROUP

TIME OF THE LATEST HEARING TEST	NUMBER OF CASES
1 - 6 mos.	159
6 - 12 mos.	59
12 - 24 mos.	55
24 - 36 mos.	14
	355

nation-wide survey of ten otologists performing mobilizations, reported that initial successes varied between 25 and 65 per cent with an over-all average of 42 per cent reaching the 30-decibel level or better. The total number of ears reported was 7400, and the average number of ears made worse was 3 per cent. The results of our first 626 cases of all degrees of deafness operated by five different members of the Winthrop Foundation team are as shown in Table I.

Table II shows the improved group of cases according to the postoperative time of the most recently recorded hearing tests. It is known that 68 ears have regressed and that 128 have maintained the improvement for a period of 6 months or longer. A large number of the 159 ears that showed an improvement between 1 and 6 months after operation were recently operated on and follow-up hearing tests at one and two years are expected to be taken at a later date. Certainly, however, some of these have regressed, and the figure of 68 ears showing regression is therefore statistically invalid.

TABLE III
TIME OF REGRESSION OF THE
IMPROVED GROUP

TIME	NUMBER OF CASES
1 mo.	14
3 mos.	30
6 mos.	12
9 mos.	1
12 mos.	6
18 mos.	3
24 mos.	2
Total	68

TABLE IV
RESULTS AFTER ONE TO THREE YEARS ON 56 CONSECUTIVE CASES
IMPROVED TO BETTER THAN THE 30-DECIBEL LEVEL

	NO. OF CASES	(PERCENTAGE)
No hearing tests at 1 yr.	10	(17.8%)
Regressed below 30 db level	12	(21.4%)
Above 30 db level at 3 yrs.	4	(7.1%)
Above 30 db level at 2 yrs.	13	(23.2%)
Above 30 db level at 1 yr.	17	(30.3%)

Table III, however, is less illusory in that it shows the recorded time of the hearing test of those ears that dropped below the 30-decibel level. It is obvious that the vast majority of closures occur within the first six months but regression may continue for as long as two years.

In an effort to show the trend of long-term results, a study was made of 56 consecutive ears from my own series that were improved by more than an average of 10 decibels for the speech frequencies and reached or surpassed the 30-decibel level.

Table IV shows that 10 ears that were not tested at one year or later are unavailable for long-term evaluation. The remaining 46

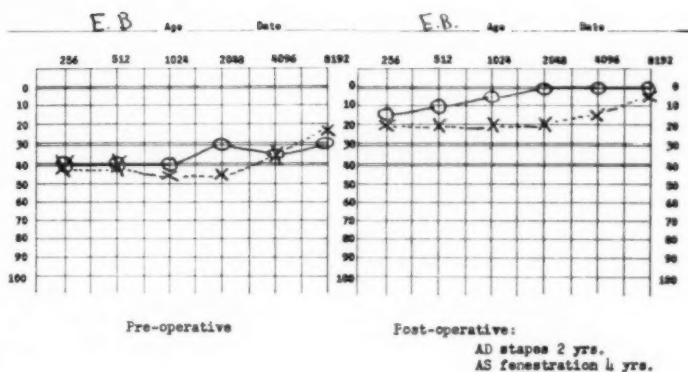


Figure 3

ears were all tested later than one year after operation and 21 per cent of them showed a drop below the practical level. Approximately 60 per cent, however, have maintained the improvement for periods of time varying from one to three years.

SUITABILITY FOR MOBILIZATION IN RELATION TO THE DEGREE OF PATHOLOGY

Observations in this series support the general contention that the ideal candidate for mobilization surgery is the one with limited, mature otosclerosis, usually in the region of the anterior footplate. The audiometric counterpart of this type of pathology is a modest hearing loss of approximately 40 decibels in the low frequencies, with a tendency for an ascending curve in the higher tones. This is illustrated in Figure 3.

Patients with more extensive involvement of the annular ligament, represented as a rule by a flat air-conduction curve, may be good candidates for mobilization, provided the bony lesion is not the active vascular type. This is demonstrated in the audiogram of Figure 4, showing the initial results of bilateral stapes mobilization.

The favorable cases for stapes mobilization are by no means limited to those patients whose audiograms are ascending or flat. One would expect the suitability for operation to decrease somewhat in

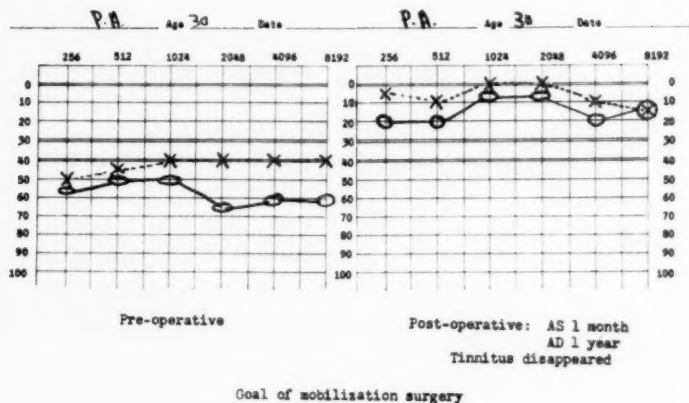


Figure 4

proportion to the descending slope of the audiogram. There were, however, so many exceptions to this general impression that it was indicated that patients should not be excluded from mobilization because of a descending audiogram. At the present time I feel that we are unable to tell what extent of otosclerotic footplate invasion is symbolic of a downward sloping audiogram. Thickening of the center of the footplate, with incomplete fixation of the annular ligament, was noted to be present in many ears with this type of sloping audiogram. Figure 5 shows the pre-operative and postoperative hearing tests on an ear with incomplete involvement of the periphery of the footplate, thicker in the center, with white, mature otosclerosis. This type of pathology may be represented by a greater loss in the high frequencies, as shown in Figure 5.

It seems that the long-term hearing result, in these types of lesions, depends not only upon the degree of trauma to the stapedio-vestibular joint during mobilization, but also on the inherent potential for progression of the otosclerosis.

Otosclerosis of the anterior footplate, if thick and more extensive, may also be treated by anterior crurotomy along with section of the footplate, as described by Basek and Fowler.⁷ Although other investigators have reported nearly normal hearing after mobilization of a small segment of the posterior portion of the footplate, this

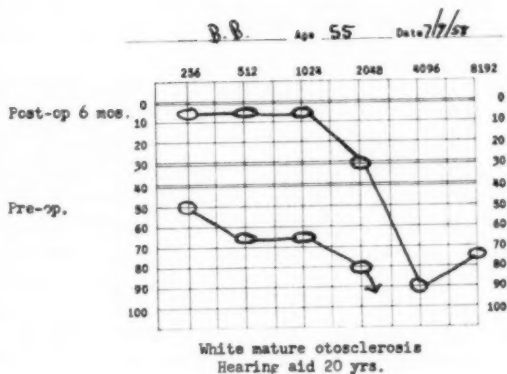


Figure 5

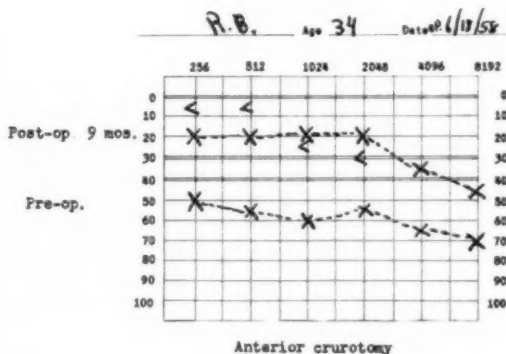


Figure 6

degree of improvement was known to have occurred in only three ears of this series, and in none did the threshold for hearing reach the 0-to-10-decibel level. Figure 6 shows the preoperative and postoperative audiogram after anterior crurotomy with mobilization of a small segment of the stapes. One would expect a disturbance of the normal leverage action of the ossicular chain with less than normal volume displacement of the perilymph when a small segment of the posterior stapedial footplate is released. The reasons for such nearly

normal hearing with the functioning of a small segment of footplate remain obscure. In general, however, the validity of the bypass concept was substantiated repeatedly throughout this series of cases.

Personal observations have impressed me with the rapidity with which regression of hearing took place after good initial results, when the otosclerotic lesion presented the picture of rough, irregular vascular bone. This represents active proliferative otosclerosis that should be treated by surgical methods other than simple fracture through the lesion. Both active and mature otosclerotic bone, of course, may be simultaneously present in the same ear. Furthermore, an apparently quiescent otosclerotic area may become reactivated at its periphery by any of the obscure factors that are responsible for the cause of otosclerosis. Nevertheless, having subjected 41 per cent of the patients to mobilization surgery without benefit, should we not ask ourselves what, if any, are the criteria that may be of preoperative value in determining the state of activity or potential activity, of an otosclerotic ear. Factors to be considered are: 1) the age of the patient; 2) the duration of the hearing loss; 3) the presence of a blush on the promontory; 4) family history of deafness; 5) the presence of stress-producing factors; and 6) the audiometric pattern.

We know that young people may have mature, isolated otosclerotic lesions, and that persons in their declining years may have extensive active otosclerosis. There appears to be no direct correlation, then, between simply the age of the patient and the state of otosclerotic activity. The age of the patient may be important if viewed in conjunction with other facts. An adolescent, for instance, with a 40-to-50 decibel air-conduction loss with a flat audiometric curve, might be expected to have areas of active otosclerosis. If, in addition, there was a strong family history of deafness, the expected long-term gain in hearing from mobilization would be unlikely.

The duration of the hearing loss may be of value in indicating favorability for mobilization. Audiometric evidence, over a period of years, that there has been little or no advance of the otosclerosis, is a favorable sign, particularly in the presence of a mild or moderate hearing loss.

The presence of a blush on the promontory suggests increased vascularity and activity of the otosclerosis and is, in general, a contra-indication to stapes mobilization. An exception to this rule would be those cases in which the annular ligament is fixed by mature bone, and the center of the footplate or the adjacent promontory is undergoing active change.

The absence of a strong family history of deafness means little, but, if present, increases the likelihood of prolonged otosclerotic activity.

Many patients with clinical otosclerosis date the onset of the deafness not only with pregnancy but with other episodes of major stress. Intensive emotional turmoil, major surgery that is accompanied by a significant blood loss, and an endocrine imbalance such as hypothyroidism have long been known to aggravate the deafness due to otosclerosis. The presence or anticipated presence of such stress factors could conceivably influence not only the time but also the type of surgery chosen.

The audiometric pattern by itself, although it may serve as a rough guide to the extent of the lesion, tells us little of the current activity or otosclerotic potential of the ear. None of these criteria for determining the degree of activity of an otosclerotic labyrinth is definitive, and since they do not lend themselves to objective measurement, the otologist can, at best, form only an intuitive estimate of the degree of activity. However, if given more than cursory attention during the preoperative examination, they will be helpful in detecting the ear in which the activity of otosclerosis precludes the likelihood of long-term hearing improvement from stapes mobilization.

OBSERVATIONS ON THE CAUSES OF FAILURE

In general the causes of failure can be attributed to one of the following factors:

1. Inadequate visibility
2. Incomplete mobilization
3. Anatomical unsuitability
4. Fracture of the stapedial crura
5. Damage to the incudostapedial joint
6. Failure to cut the anterior crus
7. Pathologically unfavorable otosclerosis

Proper positioning of the patient, meticulous control of the bleeding, and facility in manipulation of the operating microscope are paramount to definitive footplate surgery. The severing or removal of the stapedius tendon along with removal of the pyramidal

eminence is sometimes necessary to visualize the posterior crus and footplate. Any one of these minutiae may be the determining factor in the achievement of success.

Incomplete mobilization even in pathologically favorable otosclerosis is undoubtedly the most important cause when the hearing fails to reach the practical level. There is no substitute for adequate visualization of the footplate margins, although at times this may be impossible. The observation of a round-window reflex resulting from manual palpation of the stapes does not necessarily ensure maximum physiological transmission of sound.

An anatomically unsuitable oval window is one that is narrow, deeply recessed, and posteriorly placed. In this situation, if the crura are thick, only a small portion of the footplate is accessible to surgical manipulation, and if the otosclerosis is extensive, it is extremely difficult to mobilize and should be approached with a different surgical procedure. It would be helpful if one could predict the existence of an inaccessible oval window and plan surgery accordingly. I have frequently observed that the position of the long crus of the incus as determined preoperatively may offer a clue as to the anatomical disposition of the oval window. When a generous segment of the long crus of the incus is visible, anteriorly positioned, and in a vertical plane parallel with the handle of the malleus, the stapedial footplate is prone to be superficial (Fig. 7).

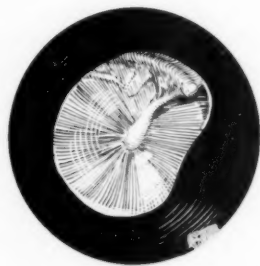


Fig. 7.—Anterior positioned incus indicating superficial foot plate.

Fracture of the stapedial crura was a most common surgical error before the use of the operating microscope. Maneuvers directly to the footplate prior to any attempts at transincudal mobilization have markedly reduced the incidence of crural fracture. In our early cases no attempts were made to reduce or replace crural fractures. It now seems feasible to accomplish this with the insertion of a prosthesis. In spite of the well-known reluctance of the stapedial crura to form callus, an attempt at replacing the fragments may at times be successful.

Figure 8 illustrates the good result after replacement of completely separated stapedial crura. This maneuver is more easily accomplished if the stapedius tendon has been cut.

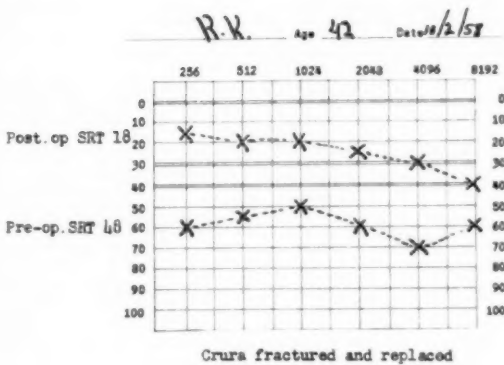


Figure 8

Separation of the incudostapedial joint will result in severe loss of hearing if not repaired. Simple reapproximation is usually sufficient. The stapedius tendon must usually be severed to allow for apposition of the articular surfaces.

Figure 9 illustrates a practical hearing gain after mobilization and replacement of a separated incudostapedial joint.

Failure to cut the anterior crus after bisecting the footplate behind the otosclerotic lesion may result in an insufficient gain in hearing. This is particularly true when the anterior crus is thick and nonresilient. With a thin, resilient anterior crus, the hearing gain in this situation may be excellent since the binding effect on the footplate mobility is very small. One of the controversial issues at present is whether or not to attempt anterior crurotomy in this instance for fear of fracturing both crura. It should be pointed out to those beginning to perform this type of surgery that anterior crurotomy is not always so easily done as one might believe from reports in the literature.

Pathologically unfavorable otosclerosis precludes the possibility of successful mobilization. It was a frequent occurrence in this series of cases and brings up the rather challenging problem of what pre-operative criteria might be utilized in order to determine inoperability. Farrior,⁸ Guilford,⁹ Bellucci,³ and others have classified otosclerosis according to the degree of pathological involvement of the stapes and

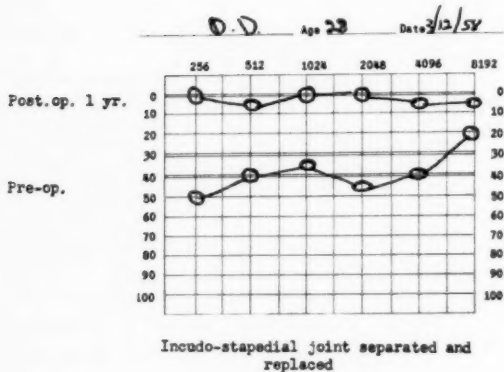


Figure 9

adjacent labyrinth. These classifications serve to point out the endless varieties and complexities that the otosclerotic pattern may assume. Obviously, one could never predict all of these specific patterns, but much information can be obtained from a study of the characteristics of the audiogram. We know that the combination of an early hearing loss and an ascending slope on the audiogram represents fixation of a small part of the footplate. We also know that massive otosclerosis of the footplate associated with involvement of the round window is indicated by a profound nerve deafness. Our criteria for prediction at the present time are untenable in regard to the majority of otosclerotic ears that fall between these two categories.

Farrior⁸ believes that bicrural otosclerosis is indicated clinically by a loss of the high tones, and Shea¹⁰ believes that a flat or early descending slope on the audiogram indicates complete invasion of the footplate periphery.

I have observed that a good many favorable results were achieved when there was a sharp slope on the audiogram, if the hearing loss for 200 and 500 cycles per second was less than 50 decibels. The pathological counterpart of this type of audiogram may be the footplate with incomplete peripheral invasion but much bony thickening in the center.

Correlation of the audiometric pattern with the location and extent of the otosclerosis impresses me as a feasible pursuit worthy of detailed study.

COMMENTS

There was a significant improvement in hearing after mobilization of the stapes in 56 per cent of the 626 otosclerotic ears in this survey. It is anticipated, on the basis of a smaller pilot study, that improvement in hearing will be maintained for periods of time varying from one to three years in more than 60 per cent of the ears.

As the gap between new surgical procedures and surgical perfection is narrowed, hope for a higher percentage of long-term good results centers around a more judicious selection of cases for stapes mobilization. This in turn depends on the correct preoperative estimate of both the extent and the activity of the otosclerosis. If the otologist knew before exploration that the pathological process would obviate both stapes mobilization and mobilization with crurotomy then he could plan to do instead the procedure of inserting a stapedial prosthesis as advocated by Shea¹⁰ and Schuknecht.¹¹ In situations in which the anatomical factors obviate both mobilization procedures and the insertion of a stapedial prosthesis, then fenestration should be planned.

243 CHARLES ST.

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XXI

PSYCHOLOGICAL CONSIDERATIONS IN THE MANAGEMENT OF CANCER

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It has been eighteen years since I was privileged to come to the Massachusetts Eye and Ear Infirmary where Dr. Leroy Schall had recently assumed his position of leadership. It is difficult not to feel that with the coming exchange of leadership, this year should be one of considerable moment to me. It is a time for sitting back, for calm reflection, for evaluation, not for Dr. Schall but for all of us who have been exposed to his stimulating and leveling influence on our developing careers.

My subject for discussion stems from exposure to an increasing number of patients with cancer, or suspected cancer, and the socio-economic environment in which they move. It concerns the when, why and what of diagnosis and treatment.

We physicians are constantly faced with decisions ranging from, "Should this gland be biopsied?"; "Should this area be biopsied or shouldn't it?"; "Should the possibility of cancer be suggested to the patient or to his family?"; "Can this lesion be irradiated without the loss of an eye?"; "Can this larynx be saved and still give as good a chance for cure by other means?" Dr. Schall has repeatedly stressed that each situation is one unto itself.

What qualifies each of us to say and do what is best for such a patient? How will the decision of management affect his family and business associates? We must meet this challenge not only as doctors, but as humanitarians as well. Our qualifications must include a knowledge of the basic psychologic needs of the individual as well as the technical skills to eradicate the organic disease.

At this point I would like to translate the remarks of one of the world's greatest physician-philosophers, Dr. Albert Schweitzer, in his analysis of the present world situation into a similar situation in the

field of cancer management. Dr. Schweitzer feels this to be the most dangerous period in history because heretofore nature has controlled man, but now man has learned to control elemental forces before he has learned to control himself. Many physicians have learned the technical skills for excising life-destroying disease such as cancer before they have developed a consciousness or philosophy of life for themselves which cannot help but be reflected in their decisions or their surgery. The objective skill of the surgeon must be tempered accordingly before he embarks on this organ-eradication decision with its many ramifications.

One who treats cancer must be a conformist to a considerable degree. He usually follows the dictates of carefully evaluated statistical evidence compiled at five to ten year intervals in Tumor Clinics which reveal the best approach to various lesions with the known existing techniques. He is a conformist in that he applies these techniques so that he may give the patient the best chance for cure or palliation depending on the type, location or the extent of the cancer in the specific individual so afflicted; that is, this same individual who also has an economic, psychologic, and moral responsibility to his family, his business associates, his employees, his community; and who has to live with himself both day and night in the months ahead.

But, cancer with its frustrating areas of location in the field of otolaryngology is not a black and white issue that can be settled on the basis of statistical past experience alone; because wherever we find it, its management seems to involve an organ of function, or the possibility of a cosmetic deformity. This, then, becomes the crux of our problem. The organ of function be it the eye, ear, nose or larynx; or the physical feature be it the auricle, facial nerve or nasal configuration; both considerations tend to blunt the surgical knife, or the radiation beam, or the cerebral cortex behind the knife or beam.

How deep to go; how wide to go; to destroy or not to destroy the ear or the eye or the larynx; how much of the nose to remove; can one really excise the tumor and save the facial nerve? What right have we to avoid destroying a facial nerve and yet take the long chance of a cure at the expense of a patient's life? How often is the attempt made to save this nerve because of the fear of the cosmetic dysfunction, only to find that residual cancer in the ear or parotid gland ultimately destroys the nerve anyway in the not too distant future; and, incidentally, ultimately destroying the patient's life as well. This isn't to condone the reckless destruction of the facial nerve, but the weight of the decision at the first surgery is tremendous.

Who gives us this almighty power to decide for this individual what is best for him? Can any of us not feel humble with such tremendous responsibility for life and death, or the prospect of a living death with intense suffering being vested in our hands and our consciences?

Well, one must be objective in his decision and face the consequences of his acts or judgment in his work as a physician as in his other worldly endeavors. Maturity is judged on the basis of how much one learns from past experience, whether satisfactory or unsatisfactory. Therefore, mature objective judgment is most essential in the management of patients with life-destroying disease such as cancer. Our main purpose is to eradicate, if possible, the disease process with a minimum of loss of organs of function and a minimum of loss of physical feature, and to restore such an individual to his place in society in as short a time as possible and as reasonably close to his former status as the nature of the disease will allow. His emotional stability should be given as much attention as possible under the circumstances; his self confidence must be restored because without it he will live in constant fear of recurrence of his disease which soon reflects itself in his family relationship, his work and the world about him. Every known method for modifying the severity of the emotional distress should be employed. Sufficient time must be allotted to the patient and his family for this purpose.

Therefore, the problem of cancer is twofold: 1) the problem of eradication or modification of the disease in order to eliminate or reduce suffering, and 2) the eradication or modification of the emotional upheaval associated with the knowledge that "I have cancer."

Let us turn to the first problem. We all aim for complete eradication of the cancer if possible, but, the possibility in each physician's hands varies with his training, his skill, his courage, his objectivity, and to some degree a sprinkling of a little wishful thinking. Be that as it may, let none of us feel so all-powerful that we cannot profit by another opinion, or additional surgical assistance, which in some cases may mean the difference between success and dismal failure. There is no halfway approach to the management of cancer. A successful goal is our aim no matter what technique is employed.

The areas of possible stumbling in our field lie in those locations where cosmetic deformity of varying degrees might be avoided. The risk of such deformity is important, but not all important if there

is a reasonable chance for cure. If there is not a reasonable chance for cure, then the possibility of great physical deformity plus the attendant emotional and physical suffering becomes a serious road-block to the management of that particular lesion. For example, if the disease involves the soft tissues of the cheek, the upper lip, the root of the nose, the ethmoid region and the orbital fascia, should one do a hemi-facectomy inclusive of all the tissues grossly involved and a wide margin beyond it? Is this mutilating operation with its very remote chance for cure—possibly 1 per cent—indicated in such a patient? Even if palliative x-ray therapy is used, the same problem in all probability will return at a later date with a recurrence of the disease. Should this patient then receive no surgical chance for cure knowing full well his desire and love for life? The choice of which therapy is best suited for this particular individual at a specific point in the disease, points up the need for a deep philosophical evaluation on the part of the physician in charge.

If a cancer of the ethmoid sinuses or antrum reaches the orbital fascia and it cannot apparently be resected completely (and not very many can be) then the best chance for cure lies in the exenteration of that orbit and its contents en bloc, if possible, with the rest of the nearby tumor. Yes, one gets away with not doing the orbit on occasion, but the saving of an eye in most instances may mean a loss of a life and the eye as well in the not too distant future. True, no one likes to remove a normal eye if it can be saved, but if in real doubt as to the extension of the disease to the orbital fascia, the most profound judgment rests heavily on the surgeon managing such a problem. It is my conviction that the 27.5 per cent five-year cures for cancer of the antrum reported by Dr. Schall in 1945 will be improved upon with greater utilization of the technique of total maxillectomy and exenteration of the orbit propounded by Dr. Harold Tabb in such cases. It is emotionally distressing to many a surgeon to remove the normal eye because he feels that with a little more knowledge of just where the cancer extended he might save it, and who knows what may happen to the other eye in the time ahead. This is a moment for objectivity for the surgeon, but the objectivity should not mean implied disregard for a very vital sense organ and eventual physical deformity caused by the loss of such an organ. Prostheses and skin graft techniques are improving all the time, and eye and maxilla depressions have been restored with sufficient success and increasing success to make such a procedure worthwhile.

Let us turn to another area. If a cancer of the ear involves the concha within 1 cm of the external meatus and possibly the under-

lying tissues, and its complete eradication means a modified radical mastoidectomy and removal of most of the auricle, it should be done. We have already shown that 85 per cent of such patients will die of the disease in ten years or less unless so treated. Removal of the auricle is a physical deformity, but it can be lived with. Plastic prostheses to replace these auricles have been fashioned so beautifully of late that they can be worn without too much trouble, and without too great an alteration in the appearance of the patient. Plastic procedures might also be employed with great benefit. To do less surgery than is required just to save an auricle could mean death on that basis, and does mean death in so many of these patients.

Let us go to the larynx. If a small cancer of a freely movable cord treated either by radiation or by laryngofissure will give similar 87 to 92 per cent five-year cures, then why should a vocal cord be removed with a resultant speech disturbance in most instances? True, there are complications from radiation which may, in a very small percentage, result later on in such edema and fixation of the cords that a permanent tracheotomy and even total laryngectomy may become essential for life; but with laryngofissure there is a much higher percentage who have permanent dysphonia. Yes, it is true, surgical extirpation is a treatment of short duration and such an individual can return to his family and society sooner, but with his husky voice that is frequently poorer in quality than before surgery. He can also return to his position or business and obtain greater immediate economic advantage than if he were forced to give up more time from his work were radiation the form of therapy employed. The radiation treatment, however, is now much less distressing to a patient because of the super-voltage rotational techniques and lack of the formerly observed skin breakdown. This treatment takes five to six weeks and keeps many of the patients away from their business and also from social contacts toward the end of the treatment. If the patient lives at some distance from the point of treatment, he is away from his home and business practically all this time. These patients often prefer not to be exposed to their associates at this time, because of their own fears of possible impending death as well as the pity expressed awkwardly by others. However, the voice returns much closer to normal in the irradiated patient and attention to their plight by others becomes less evident with the passage of time than in those who have had laryngofissure.

However, by virtue of the location and extent of the disease in the larynx, and on the basis of past statistical evidence, if surgery is believed to be by far the preferable form of therapy, then the loss

of an organ of function and physical external appearance becomes of lesser importance, and this radical surgery must be done. Total laryngectomy has become a common procedure for extensive cancer of the larynx in all the major centers of the world. Dr. Schall has been one of its earliest advocates and has recommended this procedure for definite specific reasons such as the fixation of a cord, involvement of the ventricle and false cord, subglottic extension of the tumor, and so forth. This radical procedure, despite its many personal and environmental ramifications, has been a life-saving measure for thousands of people. Many physicians and patients who have less knowledge of the wonderful end-results with total laryngectomy, tend to raise more questions about such a procedure being done than they would about total gastrectomy for cancer of the stomach. Yet, total laryngectomy has cured considerably more patients with cancer of the larynx than the gastrectomy has for cancer of the stomach. However, we are dealing with the loss of an important organ of function which becomes obvious to all the patient's contacts. Therefore, a word of caution must be introduced. Total or partial laryngectomy should not be advised indiscriminately just because the diagnosis of cancer of the larynx has been made. The quick eradication of the disease surgically makes more sense to some physicians than by the slower, less dramatic method of radiation. Mature considered judgment once again must be utilized and decisions modified if necessary to meet the individual requirements in all such instances.

Many attempts have been made to do hemi- or partial laryngectomies. It has been advocated by Ogura, Som, Pressman, Alonzo and others with the idea of saving such larynges. This can be very good, and it can be very bad, depending upon the judgment and the skill of the surgeon. The extent of the disease in a particular patient is often difficult to evaluate and the decision as to the preferred choice of therapy rests in our hands.

Wherever it is possible, let us save any organ of function such as the larynx to avoid such deformities as the permanent tracheotomy. However, the compromise should never be made if there is a limited chance for success. The first operation for cancer should always be the best operation to eradicate it. That is so well known to all of us.

We all know the tremendous help radical neck dissection has been to us in saving countless numbers of patients who might have died as a result of regional metastatic gland involvement with its eventual, miserable exitus. Bilateral neck dissections have also been of tremendous help in still fewer cases. The physical deformity in

unilateral neck dissection is very little compared with the accomplished results, but the physical deformity of the bilateral neck dissection plus the possibility of cerebral vascular changes that may produce hemiplegia and invalidity is a more serious problem. Considerable thought should be given the great possibility that the limited chance given the patient for eradication of widespread metastatic cancer by bilateral neck dissection does not later make the patient's future a living death, and the patient's family environment one of an active nursing home with its physical, emotional and financial strain. The decision to do such a procedure has many ramifications far and beyond the technical skill in the operating room.

The scope of this paper could include discussion of hemimandibulectomy and neck dissection, excisions of portions of the nose and many other procedures which carry with them considerable number of social problems not only for the patient, but for his family; but it would be merely repetition.

This discussion so far might imply that I do not favor radical surgery, but this is not so. I advocate and urge it in every instance where there is a reasonable chance for cure despite the necessity of eliminating an organ of function or producing a cosmetic deformity. But, I always keep in mind the possible innumerable ramifications involved in my decision to do or not to do a particular procedure for a specific tumor in a specific location in a specific individual, a member of a specific family unit in a specific community.

Let us briefly turn to the emotional considerations in the patient with cancer. The cancer patient when informed of his problem undergoes an understandable immediate depression; and then, in order to face reality, he attempts to adjust by developing a new attitude toward life. Many cannot and never do adjust. For those who try, it is a tremendous undertaking. Some are so shocked and numbed that they feel detached from everything about them, for some time. Others entertain suicidal thoughts which are fortunately of short duration. As the procedure for excision of the cancer and the body's altered functions are explained to him by his physician, the patient certainly passes through a phase of bewilderment. To each individual there is a different cultural or physiological or psychological factor which determines the significance of this greatest-of-all events in his lifetime. It is at this point that the male or female's healthy partner can help or obstruct the road to successful adjustment. If there has been tension and conflict of some moment between husband and wife prior to the excision of a cancer of a larynx by total laryn-

gectomy, the situation may worsen considerably due to the sudden poorer communication between them, both psychological and physical. The healthy member may either mellow and become overconsiderate, or as we so often see, the relationships may become more strained. Tension, anxiety, guilt feelings manifest themselves in both partners; resentment increases; covert hostility may become overt. Since most total laryngectomies are in men, the female partner becomes the most important part of the physician-patient-family team. It has been wonderful to see how the women have come through in most instances. They truly have shown heroic qualities. However, the few that do not, have entered a miserable existence almost equaling that of the unfortunate laryngectomee. Prior to a laryngectomy, discussion by the surgeon with the wife of a husband's real needs, his emotional problems, his future battle to be once again recognized as a whole man, and not a part-man, is so valuable to prevent a breakdown of relationships. The total laryngectomee is quite worried about his family's response to surgery. He interprets the loss of his voice box as something which has reduced his value and his acceptability by his family. His dignity has suffered a severe blow. His fear of social rejection is a real one. Despite the wonderful work done by people like Mrs. Doehler with esophageal speech therapy, how many employers, and that includes hospital administrators as well, are so willing to employ these people? The social prejudices carry over into our realistic life more than one would like to think. To many people, including some physicians, cancer is a "dirty disease." It is still considered a mark of disgrace to many lay people who must be educated to the contrary. The fear of loss of earning power and economic insecurity is perhaps the most serious that the healthy spouse must help solve. This in itself provides more fuel for the fire in the patient's soul producing more grief and guilt. The fear of death from the disease overrides these fears, but somehow most patients seem to sublimate it fairly well. The immediate anxieties of the present, the realities of day-to-day living are enough to push any one of these patients into a well of despair. We physicians must recognize and appreciate these feelings in our patients much more often than we have. One could compare equally the loss of the larynx with the loss of an eye or ear or a nose, but to a lesser extent. We live in a world of closer and closer communication and any interruption in this chain of communication, like the loss of an organ of communication, can set off a reaction of psychological responses that can be tragic.

In conclusion, we physicians whose duty, obligation and responsibility it is to eradicate any cancer to the best of our ability must

reflect, evaluate, and consider the best plan of attack on this cancer with our most mature judgment. We must consider solemnly the probable necessity for the elimination of an organ of sense or function, and or the production of a severe cosmetic deformity as a result of such a plan. We should consider not only these tiny wildly growing cells, but the body and soul of the patient, his family, his neighbors, and his associates; and then ask ourselves, is this what I would want done for me or mine?

60 CHARLESGATE WEST

OSTEOMA OF THE FRONTAL SINUS

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Osteoma of the frontal sinus is not too rare an otolaryngological disease. J. H. Cheldrey,¹ in a study of 3510 cases, reported that there is evidence of an osteoma of the sinuses in about one out of 350 routine roentgenographic sinus examinations. Of the paranasal sinuses, the frontal is the most frequent site of osteoma. In a series of nearly 300 osteomas, reported in various early twentieth century literature, 80 per cent were in the frontal sinus, 16 per cent in the ethmoid sinuses and 40 per cent in the antrum.

The disease is more common in the male and most frequently occurs during puberty.² No satisfactory pathogenesis has as yet been offered. The most prevalent belief is that the osteoma is a benign mesenchymal neoplasm, developing as a result of metaplasia of connective tissue with the formation of bone by the sinus mucosa acting as periosteum.³ Other theories are that the osteoma is initiated by an inflammatory stimulus,^{1,4} by trauma to the frontal sinus,⁵ embryonic cartilaginous cells at the junction of the ethmoidal and frontal bones,¹ and by ossification of sinus polyps. G. Keleman,⁶ reporting four cases in 1939, noted a similarity to otosclerotic bone.

Osteoma are classified as compact, cancellous or mixed.³ The compact osteoma is an ivory-hard tumor, probably formed from periosteal osteoblasts. It is limited to the skull. The cancellous osteoma possesses its own epiphyseal cartilage by virtue of which it grows as long as does the skeleton. The frontal sinus osteoma is the most commonly of the mixed type, the spongiosa forming the core.

The many suggested sites of origin are as varied as the theories of pathogenesis. In general, the osteoma may arise anywhere in the frontal sinus and is usually covered by mucus membrane.^{2,3,4,8} The most frequent site of origin would seem to be between the frontal and ethmoid bones.

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SIGNS AND SYMPTOMS

Cases of very small osteomas of the frontal sinus with no apparent impingement, but causing local pain or headache, have been published. These symptoms have been relieved by surgical removal of the osteomas.

The direction taken by enlargement governs the character of the symptoms. Increased intrasinus pressure produces frontal neuralgia and sensitiveness over the growth. The most frequent direction of enlargement is towards the orbit. This is as would be expected for this is the direction of least resistance. Diplopia results from a downward and outward displacement of the orbital contents. In its abnormal position, the eye becomes prone for secondary infection.⁴ Proptosis may occur.

The interfrontal septum may be displaced or destroyed by pressure erosion. The opposite frontal sinus is thus in this manner obstructed, infected, or involved with mucocoeles.

Growth in the zygomatic and temporal direction is not common. External deformity is quite apparent here.

Extension of the osteoma downward into the nasal cavity has been reported. Nasal blockage and discharge develops. Advancement of the tumor into the cranial fossa, either by way of the posterior wall of the frontal sinus or the cribriform plate,⁹ can produce a variety of symptoms. Headache, mental dullness, vertigo, nausea, and vomiting are early symptoms of this complication. Papilledema may be found. Hemiplegia, epilepiform seizures and pneumocephalus have been reported as more serious complications. The pneumocephalus is prone for cerebrospinal fluid rhinorrhea, meningitis, and brain abscess.

TREATMENT

Small osteomas, not posing immediate surgical problems, should have the rate of growth observed by periodic roentgenographic examinations. As soon as there are symptoms to warrant a diagnosis, the treatment should be immediate removal.¹⁰ I think this will be agreed to by all and the big question is—"What approach do we use for the removal of the frontal sinus osteoma?"

Small osteomas have been removed by an external approach through the floor of the frontal sinus without creating a defect

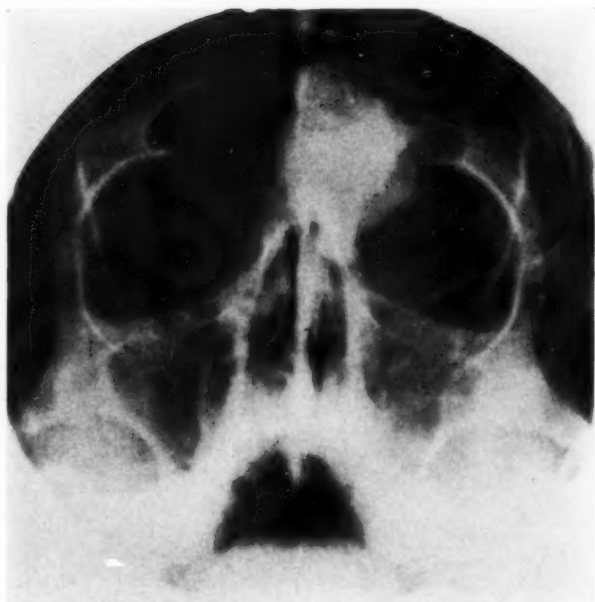


Fig. 1.—Case 1. Large osteoma in the left frontal sinus.

(Lynch frontal sinus operation). Unfortunately, most osteomas of the frontal sinus have obtained considerable size before producing the symptoms for their diagnosis.

I mention the transcranial route only to reject it as an unnecessary surgical detour.

The radical Riedel² operation (i.e. removal of floor and anterior wall) has most widely been used for removal of frontal sinus osteomas. The resultant deformity is severe enough to warrant plastic surgical repair. The Lynch and Riedel operations are also known for subsequent mucocoele and pyocoele formation. In 1950, Colver¹¹ described a "lid operation" to remove an osteoma of the frontal sinus. He drilled multiple holes and connected these with a chisel.

Dr. Zovickian¹² presented a case in 1957 in which the osteoma was removed through an anterior wall lid. He then packed the sinus



Fig. 2.—Case 1. Postoperative film. Note site of origin of osteoma and outline of osteoplastic flap.

with bone chips, before replacing the lid. There was no postoperative deformity.

I have used the osteoplastic approach to remove two large osteomas of the frontal sinus. These frontals were not obliterated with fat because of widely patent nasofrontal ducts in both cases. An outline of this operation, in some detail, follows.^{13,14}

PREOPERATIVE CARE

1. X-rays of the sinuses are taken to determine the extent of the disease and the size of the sinuses. A pattern of the sinus can be cut out from the Caldwell view and sterilized for use during the operation. This will be explained later.

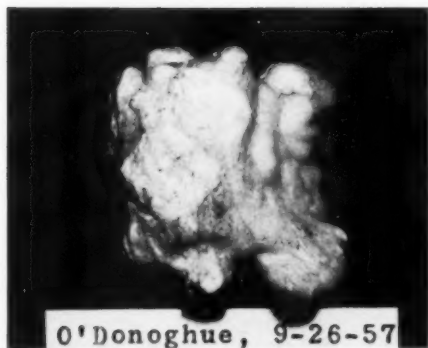


Fig. 3.—Case 1. Dense or compact osteoma.

2. Preoperative cultures of the nasal cavities are taken and if pathogens are found, then sensitivity tests are carried out.

3. The abdomen is shaved and scrubbed with phisohex solution.

Preparation of the Patient. 1. The patient's face and abdomen are prepared with phisohex or soap and surgical Prep. solution.

2. The eyelids are sutured with 5-0 dermalon.

3. Intravenous anesthesia with intratracheal tube and a pharyngeal or postnasal pack. The anesthetist is at the patient's side, as far towards the feet as is possible.

4. The area of incision is infiltrated with 2 per cent procaine or xylocaine with epinephrine.

The Incision. The incision is made along the upper margin of the eyebrow, along its full length. This incision differs from the usual frontal sinus incision in that it is not carried to bone. It thus must be made in layers to the periosteum. This is imperative, for if the blood supply to the periosteum is jeopardized, the operation cannot be carried out.

When the bleeding has been controlled, a plane of cleavage is established between the frontalis muscle and the periosteum of the frontal bone.



Fig. 4.—Case 1. Patient one and a half years after operation.

The undermining should be far beyond the limits of the frontal sinus—so that when retractors are inserted, the entire front face of the frontal sinus is exposed.

The Periosteal Incision. There are three methods to outline the periosteal incision.

1. Using a ruler or calipers, having measurements taken pre-operatively from the x-rays of the sinus.
2. A cut out from the Caldwell view of the frontal sinus, which has been sterilized, or:
3. Transillumination, at the time of operation, when the bones are not thick.

I prefer the cut-out from the sinus x-ray. The periosteal incision is made with a number fifteen blade along the sinus outline. This is carried to, but not beyond, the supra-orbital rim.

Cutting the Bone Flap. We have used the Stryker saw for the bone work. It is safe, speedy, simple, and atraumatic to soft tissues.

Following the outline of the periosteal incision, the anterior wall of the frontal sinus is cut through on a bevel. This is carried to and through the supraorbital rim.

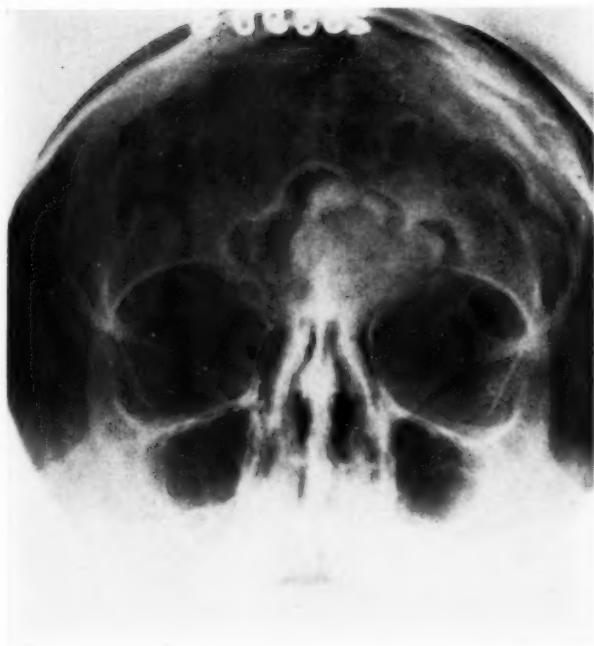


Fig. 5.—Case 2. Large osteoma left frontal sinus with mucocoele formation.

A chisel is then inserted superiorly, and the bone flap is elevated. It fractures and thus hinges along the thin floor of the frontal sinus, just medial to the supraorbital rim.

Removing the Disease from the Sinus. The osteoma is either removed intact or in fragments. The diseased mucous membrane is removed from the sinus in the usual manner with curettes and forceps. The entire lining is then further burnished with a cutting olive-shaped burr. This dissection must also include the orifice of the nasofrontal duct and the inner aspect of the bone flap, if a fat implant is to be used.

The Fat Implant. (Used when the nasofrontal duct is narrow or diseased).

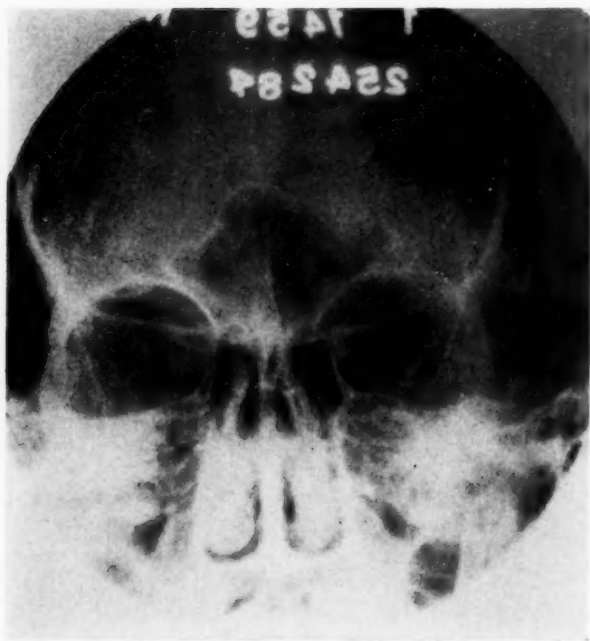


Fig. 6.—Case 2. Postoperative x-ray. Note the osteoplastic bone flap outline. The site of origin of the osteoma cannot be seen.

After a gown and glove change, the surgeon obtains a block of abdominal fat with attached rectus fascia, through a right or left rectus incision.

The vessels are tied with catgut and the skin incision closed with silk or dermalon.

The fat is trimmed to and about the size of the frontal sinus. As it is inserted, the rectus fascia is placed over the nasofrontal duct.

Closure of the Sinus. The beveled bone flap is replaced, the periosteal and subcutaneous layers are sutured with 4-0 chromic catgut and the skin approximated with 5-0 dermalon.

A dry pressure dressing consisting of telfa gauze, an eye pad, gauze fluffs and an elastic bandage, is applied.



Fig. 7.—Case 2. Postoperative photograph shows a fresh scar but no deformity.

POSTOPERATIVE CARE

1. Antibiotics are continued for at least five days. If specific antibiotics have not been determined by sensitivity tests, I have used prophylactic penicillin and streptomycin.
2. The pressure dressing is removed on the second postoperative day, the skin sutures on the fifth.
3. Postoperative x-rays are taken in two weeks, six weeks, three months, six months and one year after operation.

REPORT OF CASES

CASE 1. A 37-year-old man was admitted in 1957 after one year of intermittent headache. Three months before admission, an upper respiratory infection was complicated by diplopia. There was downward and outward displacement of the left orbit. These symptoms subsided with antibiotic therapy, but returned a few weeks before his admission.

X-rays of his sinuses (Fig. 1) show a very large osteoma in the left frontal sinus which had markedly displaced the interfrontal septum to the right and was encroaching on the cribriform plate.

The osteoma was removed using the osteoplastic frontal flap technique. The sinus was not obliterated by fat because of a widely

patent nasofrontal duct. Note the site of origin in the postoperative film and also the outline of the osteoplastic flap (Fig. 2). This is a dense or compact osteoma (Fig. 3).

A photograph (Fig. 4) shows the patient one and one-half years after the operation.

CASE 2. A 21-year-old girl was admitted to the Massachusetts Eye and Ear Infirmary in January of 1959, with a ten-year history of frontal headache. During this time, she had been examined in various clinics and by private physicians without a definite diagnosis.

X-rays showed a large osteoma nearly filling the left frontal sinus, with mucocoele formation (Fig. 5).

The tumor was removed by the osteoplastic frontal sinus operative technique. The mucous membrane of the sinus was removed along with the mucocoele. The frontal was not obliterated with fat because of a widely patent nasofrontal duct.

Postoperative films show a clear frontal sinus (Fig. 6). The site of origin cannot be made out. This osteoma is classified as the mixed type. Note the osteoplastic flap outline.

Photographs taken shortly postoperatively show a fresh scar but no deformity (Fig. 7).

SUMMARY

1. The pathogenesis, a classification, sites of origin, signs and symptoms and treatment of osteoma of the frontal sinus are discussed.
2. An outline of the osteoplastic frontal sinus operation is given.
3. Two cases are presented to demonstrate the advantages of the osteoplastic approach for the removal of frontal sinus osteoma.

243 CHARLES STREET

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XXIII

LARYNGEAL GRANULOMA AFTER TRACHEAL INTUBATION

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Laryngeal granuloma is a benign tumor composed of granulation tissue in which the microscope shows, also, the presence of connective tissue and all the elements of chronic inflammation. Histologically, it could be called chronic inflammatory granuloma. It is observed, occasionally, after the use of tracheal intubation for anesthesia; then it is seen as a growth located, as a rule, on the laryngeal aspect of the vocal process of the arytenoid. It is often bilateral, and may become large enough to obstruct the laryngeal airway.

Clausen,¹ in 1932, was the first one to report a case of laryngeal granuloma following intubation for anesthesia. The patient was a woman in whom the laryngeal symptoms, hoarseness in particular, appeared three or four weeks after the operation. However, the diagnosis was not made until six months later, when the larynx was first examined. There was, immediately below the right vocal cord, a pedunculated mass of about five millimeters in diameter. There was no time to do any treatment because the growth disappeared spontaneously after a few days, with complete relief of the symptoms.

This report of Clausen is important, not only because it is the first one, but also because it reveals two of the characteristics of traumatic laryngeal granuloma: the tendency to become pedunculated and the possibility of spontaneous cure by progressive constriction of the pedicle.

The purpose of this paper is to report three cases of laryngeal granuloma that I have seen during the last thirteen years, and to try to answer some of the questions that come to the mind when we have to deal with this unusual complication of endotracheal anesthesia.

REPORT OF CASES

CASE 1. A white man, 32 years of age, Protestant missionary, was seen in Guatemala City in September, 1946, complaining of

hoarseness and unproductive cough of three weeks' duration. Two months previously he had undergone a cholecystectomy under general anesthesia. On the posterior third of the right vocal cord there was a little tumor of, approximately, 6 mm in diameter, sessile, attached to the free border and superior aspect of the cord; the color was dark reddish. The left cord was normal. As long as the operation had been performed in a hospital of the United States, I had to wait several days before learning the details of the anesthesia. Ether had been used, with induction of nitrous oxide; a 32 Fr. rubber tube had been passed through the right nostril and guided into the trachea under direct laryngoscopic vision. It had been an uneventful anesthesia, and the tube stayed in place for 90 minutes.

In reviewing the literature, I was surprised at what little information I could find. Since Clausen's report in 1932, Gould,² Cohen,³ Smiley,⁴ Farrior,⁵ Robin,⁶ Kearny,⁷ had each seen one case. Kearny's paper describes the first bilateral laryngeal granuloma observed after tracheal intubation for anesthesia. In all the reported cases the site of the new growth was the vocal process of the arytenoid, and in all of them the tendency to become pedunculated was noted.

After studying my case for several days, I made the diagnosis of laryngeal granuloma and decided to wait, examining the patient once a week. After five weeks the mass had grown a little and had started to form a pedicle. Three weeks later it had increased in size, was definitely pedunculated, and its color had changed to yellowish. Under local anesthesia, and by means of direct laryngoscopy, the tumor was removed with relative ease. Four weeks later the larynx looked normal, and the patient's voice had gained back its original quality and tone. Microscopic diagnosis was "chronic inflammatory granuloma."

CASE 2. The second case is an Indian girl, 20 years of age, who had been suffering from pulmonary tuberculosis and had undergone a right pneumonectomy under general anesthesia. I saw her in February, 1950, two months after the operation, because the surgeon feared that she might have a tuberculous lesion in the larynx. Two weeks after the operation she had started to complain of hoarseness and a slight discomfort in the throat, which she could not describe very well. On the posterior third of each vocal cord there was a little tumor, the left one about 6 mm in diameter, and the right one 10 mm; they were opposite each other, dark red in color, smooth all around, each one attached to the free border of the respective cord. During phonation these growths came in contact, interfering with the com-

plete adduction of the cords. The left lung was clinically and radiologically healthy, and the investigation of tuberculous bacillus had been repeatedly negative. The diagnosis of bilateral laryngeal granuloma was made. I had the opportunity of speaking with the anesthesiologist, who informed me that the intubation had been very easy; a No. 6 Magill rubber tube had been passed through the nose, and guided into the trachea under direct vision. Nitrous oxide had been used for induction, and ether had been the basic anesthetic. The operation had lasted three hours and forty minutes, and the patient had been relaxed all the time.

In 1950 I was able to find better information in the literature. Tracheal intubation was used in most of the general anesthetics, and there had been reported several cases of laryngeal granuloma. There were the names of McLaurin,⁸ Tuft and Ratner,⁹ Barton,¹⁰ Finer,¹¹ Hill,¹² Wattles,¹³ in 1949, published a comprehensive review of the literature, and reported a bilateral case. New and Devine,¹⁴ in 1949 also, with the name of contact ulcer granuloma, reported 53 cases, although only 9 were observed after tracheal intubation.

All the authors agree that it is better to wait until the growth has become pedunculated, so that the removal is easier and there is less probability of recurrence. In this second case it was decided to observe the patient once a week. After four weeks examination of the larynx showed that both neoplasms had grown, being now practically of the same size; they were definitely pedunculated, and the patient showed, intermittently, signs of very uncomfortable laryngeal obstruction. Under local anesthesia and by direct laryngoscopy the two growths were removed. After six weeks the appearance of the larynx was normal; the patient's voice, also, had a natural tone. The pathological examination confirmed the diagnosis of granuloma.

CASE 3. A 38 year old white woman was first seen in February, 1956, complaining of hoarseness of sixteen days' duration. Seven weeks previously she had had a hysterectomy done under general anesthesia of nitrous oxide and ether. The examination of the larynx revealed a little tumor on the posterior third of the right vocal cord, about 4 mm in diameter, sessile, rosy in color, attached to the free border of the cord. During phonation the left cord came in contact with this mass, which did not allow the correct approximation of the vocal cords. The left cord appeared to be normal. A diagnosis of laryngeal granuloma was made. Nitrous oxide and ether had been used for the anesthesia, and the tracheal intubation had been performed with a No. 32 Fr. plastic tube passed through the

nose; the introduction of the tube into the trachea had been done under direct vision. The anesthesia had been uneventful, and had lasted 130 minutes.

I decided to wait until the mass became pedunculated, and the patient was advised to come for examination once a week. However, she had to take care of a son who had been injured and did not come until 24 days after the first visit. Much to my surprise, I did not find one granuloma, but two, one on each vocal cord. The first one had increased in size, and the other (symmetrically located on the opposite cord) was slightly smaller. Hoarseness was still the only complaint.

In 1956 the literature was more plentiful, but I could not find a case similar to mine: all the cases of bilateral laryngeal granuloma after tracheal intubation had been diagnosed as such from the beginning. Among those who had reported cases and had studied the problems of laryngeal granuloma, I found the names of Wright,¹⁵ Lieberman,¹⁶ Brown,¹⁷ Barton,¹⁸ Macneal,¹⁹ Matzker,²⁰ Peimer and Feuerstein,²¹ Ioannovich,²² Heller,²³ Myerson.²⁴ But no one had reported a case that from unilateral had changed into bilateral.

I kept the patient under close observation and examined her every three or four days. At the end of three weeks there was a slight increase in the size of the tumors. In another four weeks the growths were movable and the color had changed to yellowish. The patient started to complain of sensation of a foreign body in the throat. The masses had grown and were frankly pedunculated. During inspiration they threatened to close the glottis. Under local anesthesia and by direct laryngoscopy they were removed. Six weeks later the larynx looked normal and the patient felt completely cured.

COMMENT

In the three cases just described there was no recurrence, for they were controlled over several months. In all of them the first symptoms appeared three or four weeks after intubation; in all three the tendency to become pedunculated was observed. When there is a pedicle the removal is a simple procedure which causes little injury to the vocal cord.

Tracheal intubation has made general anesthesia far safer, and has been enthusiastically welcomed by the surgeons, too. It is used more and more every day, and the percentage of cases in which the anesthetist passes a tube through the glottis has been steadily approach-

ing 100. Consequently laryngologists are bound to find traumatic granuloma of the larynx more frequently than in the past.

Studying the reported cases, we may try to answer some of the questions that arise when dealing with laryngeal granuloma. First of all, I think that the term "contact ulcer granuloma" is confusing and should not be applied to the growth that is seen after tracheal intubation. When Jackson,²⁵ in 1928, used the words contact ulcer for the first time, he meant superficial ulcerations of indeterminate etiology; if granulomatous tissue should grow on one of these ulcerations, then the words contact ulcer granuloma describe the condition. But the growth that starts in the larynx after tracheal intubation is of traumatic origin, and I suggest that it be called "traumatic laryngeal granuloma." Contact ulcer and contact ulcer granuloma are seen almost exclusively in men, whereas a great majority of cases of traumatic granuloma are observed in women.

The mechanism by which granulation tissue starts to grow on a vocal cord after tracheal intubation has not been definitely explained. Undoubtedly, the tube traumatizes the cords at the level of the vocal process of the arytenoids, causing a little ulceration of the epithelium. In most cases the lesion does not go beyond that. Contrary to the opinion of many observers, I think that a small tube is more traumatizing than a tube that fits the larynx correctly; a small tube causes more friction to the cords because it may move more freely within the glottis. A good number of the reported cases occurred after intubation through the nose, and intubation through the nose means a small tube. Once the epithelium has been injured by the tracheal tube, the vibration of the cords during phonation may be the stimulating factor in the growth of granulation tissue. When the growth has attained a certain size, its own mass pulls from the base during respiration and phonation, forming the pedicle in a process of concentric healing.

Laryngeal granuloma, as a complication of tracheal intubation, is more frequently seen in women than in men. I think that the higher pitch of the feminine voice may be the determining factor. Why do children seem to be spared from this condition? The answer could be that in children the anesthesiologists use relatively larger tubes than in adults, and also we know how resistant the larynx of a child is to any kind of trauma, particularly abuse of the voice.

The most striking characteristic of laryngeal granuloma is the fact that it is so often bilateral. How is it possible that a complica-

tion which is so rare is so frequently bilateral? I am convinced that all starts with one small traumatic ulceration on either side; then a nodule of granulation tissue appears on one side, and the vibration of one vocal cord against the other produces the mechanical implantation of a few cells on the other side. The last case that I have presented shows the possibility of such a mechanism; I think, however, that in the majority of bilateral cases this process cannot be observed because it probably takes place before the beginning of the symptoms.

There are many unanswered questions in regard to laryngeal granuloma after tracheal intubation. For definite answers we still have to wait for the cases to be reported in the future.

15 CALLE 6-59, ZONE 1

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XXIV

POSTNASAL DISCHARGE

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This paper is concerned with the following:

- 1) Postnasal discharge of which the patient is aware is abnormal.
- 2) The cause of such a discharge can be diagnosed on the basis of the subsequent information given here.

3) This information is presented for practitioners in diverse fields, since the symptom under discussion is frequently encountered in various kinds of medical practice and commonly misinterpreted. Therefore, it is not intended as a particularly new source of medical knowledge to a specialized group except that the ensuing consideration by etiological categories may be a diagnostic help.

4) The causes of postnasal discharge may be concentrated into the unintelligible word "MMITNNS," which represents the first letter of each general class according to etiology. Poetic license or imagination allows one to sound this collections of letters as the word "MIT-TENS." If one remembers the original spelling, a mnemonic device is available which includes the first letter of all the groups of postnasal discharge.

5) I hope to include, briefly but incompletely, pathologic physiology, diagnostic features, and treatment in general. This limitation in scope should be practical for those in other fields who may have to consider this symptom.

First of all, a postnasal flow of mucus is a normal function and only when the patient becomes conscious of it does it assume clinical significance. Secondly, this symptom is seldom due to the often alleged "sinus trouble" which implies infected sinuses. Thirdly, an understanding of postnasal discharge, both normal and abnormal, depends on an understanding of the formation and flow of the nasal mucus.

One thousand to fifteen hundred cubic centimeters of mucus is formed in twenty-four hours by the mucous glands in the nose

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and sinuses.⁸ This liquid forms a film over all the mucosa and is moved in a definite direction by the co-ordinated beat of the cilia, tiny hair-like processes projecting from the epithelial cell, each of the latter containing eight to ten cilia. The mucus current in the sinuses is toward the ostia, in the nose toward the choanae, in the nasopharynx diffusely over all surfaces and then downward. The great majority of the mucus is formed in the nose, the sinuses forming just enough, under normal conditions, to moisten their surfaces and to provide a current for removing any possible foreign material. The formation of this fluid is controlled by the autonomic system, the parasympathetic causing increased production, the sympathetic acting in opposite fashion.⁶ Its normal composition is 3% mucin and 97% water. This moving liquid blanket has two main functions. The first and most important is to humidify the inspired air. Somewhat less than half of the total output is used in this way,⁸ the excess reaching the hypopharynx where it is swallowed. Incidentally, this fact is important in bulbar disease where, if the swallowing and coughing reflexes are diminished or absent, a patient can drown shortly in his own secretions. The second function is as a mechanical washer. Foreign particles, bacteria, etc., are caught in the tenacious mucus film which, moving several millimeters a minute, prevents them from settling and producing irritation or infection.

Abnormalities of mucus production, flow, or composition, giving rise to the sensation of postnasal discharge, may occur as follows:

- 1) Autonomic imbalance. This results from noxious stimuli such as allergy, infection, irritant fumes, etc. Parasympathetic excitation produces an excess of mucus.

- 2) Glandular atrophy, as seen in atrophic rhinitis and the mucosal atrophy of old age, causes diminished formation of mucus. Because of evaporation, the remainder becomes viscid, cannot be moved by the cilia at a normal rate, stagnates, and may become secondarily infected.

- 3) The ciliary beat may be slowed or stopped by the repeated use of such drugs as cocaine or epinephrine, but, of special importance, by dry air. Although temperature changes do not seem to affect the cilia, low humidities seriously interfere with their function. The importance of this ciliary paralysis and subsequent stagnation of mucus in the production of respiratory infections in the winter time is obvious.

- 4) Intranasal hard or soft tissue obstructions mechanically impede the secretory current and may initiate the sensation of

postnasal discharge by retention of mucus as well as by excessive evaporation due to the abnormal ventilation of misdirected air currents.

5) Finally, the composition of the mucus may be too thick or thin, as it is produced, lessening the mechanical efficiency of the cilia and causing delayed flow. Examples of this are the viscid mucus of hypothyroidism and the thin fluid of certain allergies.

Actually, in order *not* to have conscious postnasal discharge, a normal amount of mucus must be formed, of normal consistency, moved by normally-acting cilia, along normal anatomic pathways.

In any case of postnasal discharge, more than one etiological factor may be concerned. For example, the viscid mucus in New England noses produced by lack of humidity in the winter may cause paroxysms of "hawking" and even gagging in an attempt to dislodge a tenacious mass of material from the choanal region or the upper surface of the soft palate. The patient welcomes subsequent infection at first because the outpouring of mucus as the result of infectious stimulation makes his secretion amenable to expectoration.

On the basis of what has gone before, consideration of the following categories of postnasal discharge, based on the mnemonic "mmitnns" has proved useful to me when taking a history. Some familiarity with intranasal examination is useful in diagnosis, but it is surprising how accurate one may be with the history alone. In any case, it is well to keep two things in mind. First, consider the patient as a whole in your history, observation, and examination; and, secondly, remember that practically all infected nasal discharges, of any consequence or duration, except the initial stages of a virus rhinitis, are colored. Thus, infection whether primary or secondary, can be quickly ruled in or out by one or two questions concerning color.

Mechanical. Congenital or post-traumatic intranasal distortions and deviations, such as deflected septa or hypertrophied turbinates, interfere with normal mucus flow by causing abnormal ventilation and often obstruction. Soft tissue anomalies may do the same. Too much mucus may be evaporated if one nasal chamber is too large, rendering the remainder viscous, and its movement slowed.¹¹ Pathological eddying and turbulence, because of mechanical faults, may act similarly, even in a narrowed nostril. Jet action of misdirected air streams can dry a particular area, causing accumulation of thick

mucus here. It is only one step more to bacterial penetration and infection. The normal motion of the mucus blanket may be obstructed by mechanical faults. Piling up, drying, and slowing of the stream ensue, making the patient conscious of his postnasal mucus. Diagnosis is suggested by a chronic history of deficient nasal ventilation, one or both sides, mucoid postnasal discharge, and the presence, on inspection, of intranasal mechanical faults. Deformities of the external nasal contour are often present. Treatment is surgical correction if the symptoms are severe enough. The presence of a foreign body is included under this heading because it obstructs breathing and causes one-sided anterior and posterior discharge. Also, a postnasal discharge following trauma (mechanical) is sometimes due to a leak of cerebro-spinal fluid, usually a one-sided, watery, anterior and posterior nasal emission. Elimination of the leak, if it does not occur spontaneously, is done by intracranial surgical procedures.

Metabolic. Hypothyroidism has already been mentioned as a cause of postnasal discharge, probably due to a change in the extracellular tissue fluid.⁹ These patients have a thickened mucus, produced either by a dry, red mucosa or a pale boggy one.⁹ Hypothyroidism can be secondary to some other endocrine disturbance, such as hypopituitarism, so that awareness of the patient and his glandular system as a whole is necessary in diagnosis. A lowered metabolic rate as a cause of nasal complaints is quite common, but the patients are rarely myxedematous and the diagnosis is usually made by suspicion, exclusion of other factors, and a lowered basal metabolism or protein-bound iodine. Personally, I think a temperature chart kept by the patient at home, which averages definitely less than 98.6, is the simplest, cheapest, and most accurate way of diagnosing hypothyroidism. Treatment by thyroid is gratifying in most cases.

The excessive production of nasal mucus in pregnancy is the other common example in the metabolic group. Once developed it often persists until parturition. Therapy is symptomatic and attention to complications such as sinusitis which may develop secondary to the mucosal engorgement and interference with sinus drainage.

Infectious. The colored nature of almost all nasal discharges of infectious origin has been mentioned. This characteristic is very important. The material is most often yellow, sometimes green, occasionally brown or gray. Infection as the primary cause of postnasal discharge occurs only in a minority of cases. It may be one-sided or it may be secondary, the history revealing a prior mucoid discharge, due to some other cause contained in one of the other categories. In the presence of definitely developed infection, sinus

involvement is frequent, and transillumination or x-rays will show this. Furthermore, sinus implication is seldom symmetrical, in contradistinction to allergy. Treatment is initially medical and consists of non-irritating shrinking sprays to keep the nose, especially the regions of the sinus ostia, open in order that drainage may proceed. Antibiotics or chemotherapy are used, if necessary, as well as other medical devices such as displacement therapy, systemic vasoconstrictors, etc. Surgery is indicated only occasionally in this era of "magic drugs" and better understanding of nasal physiology. It should be done when medical treatment fails, if infection has produced irreversible tissue changes, when relief of some obstruction is necessary, or if complications threaten. Radical and extensive surgical procedures are exceptionally needed.

Toxic. The production of excessive mucus and postnasal discharge by irritating substances in the atmosphere is well-known. For example, it is found in workers in quarries, mines, or industries producing chemical fumes. Individuals who smoke to excess present the same picture. Similarly, over-medication with nose drops produces a rhinitis medicamentosa and excessive mucus formation. Ingestion of alcoholic drinks may cause postnasal discharge, presumably by the hyperemia they excite, resulting in a surplus of mucus. The commonest toxic factor in our environment, however, is dry air. As has been mentioned, it causes slowed ciliary action, viscid mucus because of evaporation of a stagnant current, accumulation of the latter, and the awareness of postnasal material. Secondary infection of this abnormal and abnormally gathering mucus is the very probable reason for the prevalence of upper respiratory infections in the winter. Therapy for toxic postnasal discharges is avoidance of the offending agent or by humidification of the air.

Neurogenic. Neurogenic postnasal discharges are the commonest because allergy is included here. The latter's parasympathetic excess produces a large amount of mucus as well as some degree of soft tissue obstruction. The abnormal arteriolar spasm of allergy is followed by slowed capillary circulation and dilatation, anoxia of capillary walls, increased permeability, augmented extra-cellular fluid content, and consequent swelling.¹⁶ Other allergists believe this engorgement is due to direct action of histamine or histamine-like substances on the capillary wall. The allergic reaction is brought about by physical, bacterial, ingested, or humoral agents. Non-allergic people would react similarly if the same stimulus were greater in degree or longer applied or both. Thus, allergy is a difference in degree really. It is a striking fact that emotional or mental aberrations

tions or stimuli can produce a picture indistinguishable from sensitivity,¹⁸ even large numbers of eosinophils being present in the mucus secretion of the nose. A third condition producing the same nasal picture is vasomotor rhinitis where no detectable sensitivity is present and the psyche is stable. The trouble is apparently an instability of autonomic innervation, and I term these cases "lower level" neurogenic ones, indicating the nervous fault to be below the cortical level. Thus, a similar nasal picture is present in true allergy, psychogenic instability as in the neuroses and psychoses, and vasomotor or "lower level" instability.

The frequency of sneezing and itching in allergy, and the tendency of the latter to be seasonal or related to certain exposures may differentiate it from the psychogenic or neurogenic case. The relationship of the adrenal cortex to the stimulus of allergy on one hand and the excitation from higher nerve centers on the other remains to be clarified. Perhaps some day these groups will merge into one.

The diagnosis of neurogenic postnasal discharge, then, depends on a detailed history, accurate skin testing, alternate elimination of and exposure to possible offending substances, trials of antihistamines, an awareness of mental and emotional states as important causes, and, frequently the help of the allergist or the psychiatrist. The patient who presents no detectable sensitivity and has a stable psyche, yet has an apparently allergic nose with mucoid postnasal discharge has vasomotor rhinitis, diagnosed really by exclusion.

Details of treatment are familiar in most respects. In the allergic case, avoidance of allergens or injections to counteract them usually works well. Antihistamine drugs are sufficient for the mild seasonal case. The occasional severe allergic episode needs ACTH or cortisone temporarily. Some authorities¹⁶ recommend a regime calculated to obviate the abnormal peripheral arteriolar spasm and capillary leakage. This consists mainly of vasodilators, cholinergic blocking agents, and measures to prevent fluid retention such as a low sodium diet and diuretics. Surgery to restore proper ventilation and drainage should be done in cases where irreversible tissue changes (i.e. polyps, etc.) interfere with these two functions. Radical exenteration is occasionally necessary. The treatment of postnasal discharge of psychic origin theoretically lies within the realm of the psychiatrist, but the otolaryngologist can achieve much by common-sense advice. In this respect, referral to the patient's priest, minister, or rabbi sometimes works wonders. Vasomotor rhinitis may possibly be controlled by appropriate autonomic drugs or the anti-spastic arteriolar regime mentioned above. In any case, intranasal cauterizations or other minor

surgical procedures to restore ventilation and drainage may be necessary if the condition has been present for a long time.

New Growth. This constitutes a minor cause of postnasal discharge. Almost any benign or malignant tumor has been found in the nose. Symptoms are usually one-sided, the discharge is frequently blood tinged, and is both anterior and posterior. Diagnosis is confirmed by inspection and biopsy. Treatment is appropriate surgery, irradiation, or both.

Systemic Disease is the final category in the mnemonic "mmitnns." Here postnasal discharge is part of a general picture. Occasionally it is predominant until the diffuse disease supervenes. Infections such as influenza and infectious mononucleosis usually have some rhinitis present, with a postnasal flow which may or may not be purulent. Circulatory disease such as heart failure or chronic nephritis with uremia and collagenous states such as lupus erythematosus are examples of systemic conditions with nasal symptoms. Treatment is that of the basic disease with symptomatic relief directed locally.

SUMMARY

1. A postnasal discharge of mucus, a continuously normal function of which the patient is not aware, becomes abnormal when it causes a subjective sensation. This may be due to an increased amount, a thickened consistency, or faults in the normal mechanics or flow.

2. This symptom is common and frequently misinterpreted. The general categories of abnormal postnasal drip have been listed according to etiology. The first letter of each group, in the order of description, spells the unintelligible "mmitnns," which imagination can interpret as "mittens." If the original spelling is remembered, a mnemonic device is thus available to remember these general classes, into one of which I believe any case of postnasal discharge can be placed. They are:

Mechanical
Metabolic
Infectious
Toxic
Neurogenic
New Growth
Systemic

3. The commonest causes of postnasal drip in each category are mentioned, the pathological physiology is discussed, and diagnosis and treatment are indicated.

507 MAIN ST.

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REAPPRAISAL OF SEVENTY-FIVE CASES
OF RADICAL NECK DISSECTION
FOR CARCINOMA OF THE LARYNX

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JAMES B. SNOW, JR., M.D.

BOSTON, MASS.

The widespread use of radical neck dissection in the treatment of carcinoma of the larynx is well established. Uncertainty exists as to the indications for the procedure. It is necessary to define the indications for this procedure by careful evaluation of the results of this therapy in specific cases. In 1956 in a study of the two year survival of 75 cases of radical neck dissection at the Massachusetts Eye and Ear Infirmary by one of us (GFR), a striking correlation was found between prognosis and the pre-operatively estimated size of the metastatic node.⁵

In a more recent study in 1959, we were impressed with the relatively poor survival rate when operation was advised to practically all patients in whom a metastatic node was found. In an attempt to find factors which would be helpful in choosing patients in whom the indications for surgery were valid, we were impressed with the importance of the site of the primary lesion, the number of nodes, and (although we were unable to demonstrate it on actual node size) the size of nodes. It is the purpose of this study to reappraise the group of 75 patients reported in 1956 on a longer term follow-up in the hope of increasing our understanding of the role of the number and size of nodes in prognosis.

The data presented are based on 75 unselected, consecutive patients who had a radical neck dissection for carcinoma of the larynx from 1948 to mid-1955. Forty-two patients had been treated by laryngectomy, 6 by laryngofissure, 25 by combined laryngectomy-neck dissection, and 2 by radiation to the larynx. There were 72

From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary and Harvard Medical School.

TABLE I
SITE OF PRIMARY LESION

<i>Cordal-Subglottic</i>	24
Cordal	20
Cordal and Subglottic	4
<i>Vestibular</i>	20
Supracordal endolaryngeal	2
Cordal and Supracordal endolaryngeal	15
Cordal, Supracordal endolaryngeal and subglottic	3
<i>Supravestibular</i>	20
Epiglottic	3
Supracordal endolaryngeal and epiglottic	9
Cordal, Supracordal endolaryngeal and epiglottic	5
Epiglottic and Base of Tongue	3
<i>Hypopharyngeal</i>	11
Hypopharyngeal	4
Hypopharyngeal and Base of Tongue	1
Hypopharyngeal and Supracordal endolaryngeal	6

males and 3 females. The patients' ages ranged from the fourth to the eighth decade with two-thirds in the 50-70 years group. Table I shows the site of the primary lesion according to the classification used and described in our recent paper.⁶ All of the patients had squamous cell carcinoma.

DATA

Table II shows an overall survival rate of 35% for potential three year survivors and 27% for potential five year survivors. Fifty-seven per cent have died of their disease and nearly three-quarters of these within the first two years. These survival figures fairly well approximate the survival figures of others.¹⁻⁴

In the 1956 study the patients were divided into two groups on the basis of the preoperative estimate of the size of the largest node. One group consisted of patients who had a node 2 centimeters or smaller, and the other consisted of patients with a node larger than 2 centimeters. In 1956 the two year survival was 87% if the node was 2 cm or smaller and only 7% if the node was larger than 2 cm. Tables III and IV show the three and five year survival data on these 75 patients on the basis of whether the node was larger or smaller

TABLE II
SURVIVAL

		PERCENT OF TOTAL
<i>Lost to Follow-Up</i>	2	2.6%
<i>Dead of Other Causes</i>		
More than 2 years	2	2.6%
Less than 1 year	1	
More than 1 year	0	
More than 2 years	1	
More than 3 years	0	4%
More than 4 years	0	
More than 5 years	1	
	3	
<i>Living With Recurrence</i>		
More than 4 years	1	
More than 5 years	0	1.3%
	1	
<i>Dead of Disease</i>		
Less than 1 year	16	
More than 1 year	15	
More than 2 years	7	57%
More than 3 years	3	
More than 4 years	1	
More than 5 years	1	
	43	
<i>Living Without Evidence of Recurrence</i>		
		% OF POTENTIAL SURVIVORS
More than 3 years	4	50%
More than 4 years	9	47%
More than 5 years	13	27%
	26	
Total potential 3 year survivors		75
Total potential 5 year survivors		49

TABLE III
THREE YEAR SURVIVAL ON THE BASIS OF ESTIMATED
SIZE OF NODE AND PRESENCE OF METASTASIS

ESTIMATED SIZE OF NODE		LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
2 cm or smaller	Positive	12	9
	No evidence of metastasis	8	5
Larger than 2 cm	Positive	5	27
	No evidence of metastasis	1	1

TABLE IV
FIVE YEAR SURVIVAL ON THE BASIS OF ESTIMATED
SIZE OF NODE AND PRESENCE OF METASTASIS

ESTIMATED SIZE OF NODE		LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
2 cm or smaller	Positive	5	3
	No evidence of metastasis	3	3
Larger than 2 cm	Positive	5	23
	No evidence of metastasis	0	1

than 2 cm. Since it was thought that the survival rate of the group with nodes 2 cm and smaller might be unduly influenced by the fact that most of the patients who were found to have no evidence of metastasis were in this group, the patients with no evidence of metastasis are presented separately. Such was not the case. If the node was 2 cm or smaller, the three year survival rate was 57% and the five year survival was 63%. If the node was larger than 2 cm, the three year survival rate was 14% and the five year survival rate was 18%.

A more detailed study of the survival rate by each individual node size is presented in Tables V and VI. Again those patients with no evidence of metastasis are listed separately. For patients with nodes 1 cm or smaller, the three year survival rate is 75% and the five year survival rate is 100%. The survival rate begins to fall off if the node is larger than 2 cm. If the node is 3 cm the three year survival rate is 27% and the five year survival rate is 33%. There

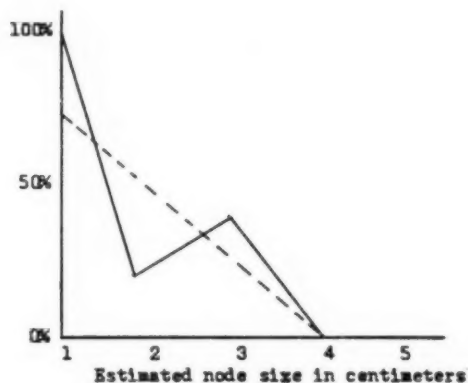


Fig. 1.—Survival rate and size of node. - - - = 3 year — = 5 year.

are virtually no three year or five year survivors with nodes of 4 cm or larger. These data are presented graphically in Figure 1.

Tables VII and VIII present the survival data with respect to the number of positive nodes found in each neck specimen. There is a marked drop in the three year survival rate if more than one node is positive. It is important to note that there are no five year survivors with more than one positive node.

Tables IX and X present the survival data on the basis of whether or not the nodes are fixed on physical examination. Only one patient in 18 survived three years with fixation of the node, while 44% survived three years with a positive movable node. Only one patient in 16 survived five years with fixation of the node while 45% survived five years with a positive movable node.

COMMENT

The original finding that the pre-operatively estimated size of the largest node affects survival appears confirmed on a longer follow-up of these 75 patients. It was found that this relationship is not based on the fact that those patients without positive metastasis had small nodes. The prognosis decreased markedly if the node was larger than 2 cm. Of those patients with 3 cm nodes, 27 per cent

TABLE V
THREE YEAR SURVIVAL ON THE BASIS OF ESTIMATED
SIZE OF NODE AND PRESENCE OF METASTASIS

ESTIMATED SIZE OF NODE		LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
1 cm	Positive nodes	6	
	No evidence of metastasis	3 9	3
2 cm	Positive nodes	6	6
	No evidence of metastasis	1 7	1 7
3 cm	Positive nodes	4	11
	No evidence of metastasis	1 5	
4 cm	Positive nodes		6
5 cm	Positive nodes		7
6 cm	Positive nodes		2
7 cm	Positive nodes		1
10 cm	Positive nodes		1

TABLE VI
FIVE YEAR SURVIVAL ON THE BASIS OF ESTIMATED
SIZE OF NODE AND PRESENCE OF METASTASIS

ESTIMATED SIZE OF NODE		LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
1 cm	Positive nodes	3	
	No evidence of metastasis	2 5	
2 cm	Positive nodes	1	3
	No evidence of metastasis	1	1 4
3 cm	Positive nodes	4	8
4 cm	Positive nodes		4
5 cm	Positive nodes		6
6 cm	Positive nodes		2
7 cm	Positive nodes		1
10 cm	Positive nodes	1	

TABLE VII
THREE YEAR SURVIVAL ON BASIS OF
NUMBER OF POSITIVE NODES

NUMBER OF POSITIVE NODES	LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
1	12	17
2	1	6
3	1	4
4	1	4
5		3
7		1
8		1
10		1
14		1

TABLE VIII
FIVE YEAR SURVIVAL ON BASIS OF
NUMBER OF POSITIVE NODES

NUMBER OF POSITIVE NODES	LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
1	9	11
2		4
3		2
4		3
5		3
8		1
14		1

survived three years and 33 per cent survived five years. The striking point is that there were virtually no survivors if the node was estimated as 4 cm or larger. In view of the fairly good five year survival rate of patients with 3 cm nodes, it appears that our previous emphasis on a node size of 2 cm was misplaced. This study would indicate that a sliding scale between 2 and 4 cm is more apropos. When the node is smaller than 2 cm the prognosis is good, whereas if the node is 4 cm or larger the prognosis is very poor, with a rapidly decreasing prog-

TABLE IX
THREE YEAR SURVIVAL ON BASIS OF FIXATION OF NODE

STATE OF FIXATION OF NODE		LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
Fixed positive node		1	17
	Positive node	16	20
Movable			
	No evidence of metastasis	5 21	1 21

TABLE X
FIVE YEAR SURVIVAL ON BASIS OF FIXATION OF NODE

STATE OF FIXATION OF NODE		LIVING WITHOUT RECURRENCE	DEAD OF DISEASE
Fixed positive node		1	15
	Positive node	9	10
Movable			
	No evidence of metastasis	2 11	1 11

nosis between these two node sizes. Our original premise regarding the importance of discovering and removing nodes by neck dissection when they are smaller than 2 cm is borne out by this study on a five-year survival basis.

Despite the much better prognosis in smaller nodes, it must be kept in mind that some of the forty-three patients who are reported in this series as dead of disease may have had truly useful palliation. In selected cases, radical neck dissection may be indicated even in large fixed nodes where the chance of "cure" is remote.

The data confirm the importance of multiple metastasis to the neck in lowering the prognosis. Certainly fixation of the metastatic node adversely affects prognosis.

It is not our opinion that the size or number or mobility of a metastatic node in any patient contraindicates radical neck dissection, but they should be seriously considered in deciding whether to advise

neck dissection in patients whose nodes have grown numerous, large, or fixed to underlying structures. We are of the opinion that the greatest benefit of radical neck dissection is available to the patient with the small, single, freely movable node and wish to urge, not fewer neck dissections, but rather more early neck dissections wherein the greatest hope of cure exists.

243 CHARLES ST.

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XXVI

AN IBM RECORD CARD FOR STAPES SURGERY

JOHN J. SHEA, JR., M.D.

MEMPHIS, TENN.

Recording and analyzing the important data in otosclerosis surgery becomes very difficult when a large series of cases is collected. I have devised an IBM record card to facilitate this.

The card measures $8\frac{1}{2} \times 11$ inches and is printed on both sides in two colors. It is divided into 77 vertical columns with 12 spaces in each column, a total of 924 spaces. X-marks are placed in the appropriate spaces. After the data have been recorded these cards can be analyzed in two ways: Directly, by going through and sorting out those with X-marks in the spaces you are interested in, and indirectly, by having IBM data processing cards punched, which can be electronically sorted. This is done by the Business Service Corporation which is a subsidiary of IBM with offices located in major cities of the United States and abroad.

DESCRIPTION OF THE CARD

Patient Information Questionnaire (Columns 1 to 11). Usual information of the patient's history.

Hearing Test (Columns 12 to 43). 1. Air and bone conduction for both ears.

2. Per cent of phonetically balanced words for both ears.

3. Speech reception threshold for both ears.

4. Patients divided into 4 groups according to the average bone conduction for the three speech frequencies:

Shambaugh groups:

A. 15 db or less

B. 16 - 25 db

C. 26 - 35 db

D. More than 35 db

Surgical Information (Columns 44 to 57). 1. Anatomy and pathology observed, especially the condition of the stapes and location of the otosclerotic focus, since these influence the outcome of the surgery.

2. Initial and final mobility, together with surgical maneuver used, since it is important to correlate the results obtained with the exact procedure used. There are several open spaces under surgical maneuver so that any special operation can be recorded by writing it in.

3. Complications, immediate results, hearing gain on the table, prediction of outcome and exact diagnosis are recorded in detail to evaluate the results obtained and to direct further surgery when necessary.

Postoperative Follow-up (Columns 58 to 77). 1. Analysis at 3, 6, 12 and 24 months of the average of the three speech frequencies for these four criteria:

- a. Air conduction decibel gain.
- b. Hearing: did it reach the 30 decibel level?
- c. Decibels of air-bone gap remaining in the operated ear.
- d. Percentage of improvement $1/4$ (25%), $1/2$ (50%), $3/4$ (75%), $4/4$ (100%) determined by:
db air conduction gain
db preoperative air-bone gap

2. Patient number.

In addition, there is a blank space for any important special information to be written.

SUMMARY

With this card the important information in otosclerosis surgery can be recorded in such a way that it can be easily analyzed according to four suggested criteria.

Abstracts of Current Articles

EAR

The Relation of Endemic Goiter to Deaf-Mutism

Greenwald, Isidor: A.M.A. Arch. Otolaryng. 70:541-44 (Nov.) 1959.

In an earlier paper, the author pointed out that deaf-mutism had been observed associated with goiter as early as the 1820s. While the relationship between endemic goiter and deaf-mutism was well recognized by Bailarger (1873) and Bircher (1883), since that time the subject has almost disappeared from the literature on goiter. Recent thyroid references tend not to mention deaf-mutism or to classify it as a manifestation of cretinism. In this paper the author has gathered data on the prevalence of goiter, deaf-mutism and cretinism and he finds that these data indicate that, in regions where goiter is common, deaf-mutism is *not* dependent upon cretinism. He noted differences in the relationship between goiter and deaf-mutism in different countries and is of the opinion these differences are real ones and worthy of consideration in assigning etiologic factors to these conditions. For example, he considers that his data add to the evidence that endemic goiter is not due to a lack of iodine but to some other agent, or agents, that need not be the same in all places at all times.

The author draws upon statistical studies which were done in various years from 1864 to 1886, except those for the U. S. and Canada, which were done in years ranging from 1910 to 1931 and the study in Peru, which was done in 1940. Specifically, a large number of deaf-mutes was found in those parts of Peru and Argentina where goiter was most prevalent—in these areas the number of deaf-mutes greatly exceeded the number of cretins and idiots. No such high proportions of deaf-mutes (5/1,000) as found in these areas has ever been reported from similar sized or populated territories. By contrast, in an area of France where goiter was four times as frequent as in the studied areas of South America, the proportion of deaf-mutes to the total population was only one-third as great. In another area of France, with 9.2 per cent goiters, the proportion of deaf-mutes was similar to that in goiter-free regions. In three of the most goitrous areas, cretins and idiots significantly outnumber deaf-mutes. In Switzerland, in the five cantons with the greatest proportion of deaf-

mutes, these people outnumbered those afflicted with mental disease. Also, figures showing the proportion of young men called for military service and rejected for goiter, when grouped for study, showed obvious correlation between rejections for goiter and proportion of deaf-mutes. In another Swiss district, close correlation was found between figures for goiters, deaf-mutism and cretinism; in goitrous communities the number of deaf-mutes was greater than that of cretins. The number of deaf-mutes/100,000 population in several states in the U. S. and in several provinces in Canada, showed no significant differences between "goiter belt" areas and those immediately contiguous to the ocean. The highest figures were comparable to the lowest in Peru and Argentina and were lower than those for all but a few of the cantons reported in the studies of Switzerland.

HILDING

Inner-Ear Mechanics and Deafness. Special Consideration of Ménière's Syndrome

Lawrence, M., and McCabe, B.F.: J.A.M.A. 171:1927-1932 (Dec. 5) 1959.

Within the inner ear, a loss of hearing may result from poor mechanical transmission of the sound vibrations, or depression of the sensory cells from a toxic substance in the endolymph. Both effects may be present in the endolymphatic hydrops of Ménière's syndrome. The authors dispute the theory that hydrops increases the endolymphatic pressure enough to interfere mechanically with sound transmission, and point out that cochlear potentials are unaffected by pressure changes. The toxic effect of an unknown chemical also would better explain the precipitous attacks.

They present the temporal bone histology in a 43-year-old woman dying following cardiac arrest prior to destructive labyrinthotomy for Ménière's. The cochlear duct showed typical endolymphatic hydrops with distention of Reissner's membrane into the scala vestibuli. Of especial interest was the evidence of rupture and repair of the membrane. Similar changes of less degree were found in the saccule.

They suggest that the increasing quantity of endolymph causes Reissner's membrane to bulge in the early stages, equalizing pressure. Eventually rupture does occur mingling peri- and endolymph, chemically affecting the sensory cells, but not histologically detectable. Repair of the rupture improves symptoms. The walls of the saccule, next in thinness to Reissner's membrane, next show the effects of

increasing pressure. The utricle and semicircular canals are rarely and much later affected. If the hydrops cannot be controlled medically, early destruction of the cochlea seems more logical than late labyrinthotomy; this attacks the compartment in which the disease arises and might preserve vestibular function.

TRIBBLE

Registration of the Stapedius-Muscle Reflex, Cutaneously Elicited, by Means of Impedance Measurements of the Ear

Klockhoff, I. H., and Anderson, W.: Arch. Ohr., Nas., u. Kehlk.-Heilk. 175: 484, 1959.

Impedance measurements of the human ear can be used to demonstrate the reflex action of the stapedius muscle. According to Metz (1951), failure to elicit this reflex in ears with impaired hearing indicates an impairment of middle ear function.

In 1956, one of the authors (Anderson) had developed a new method for measuring the impedance of the human ear: Instead of employing the acoustic bridge of K. Schuster as done in most earlier methods, the relative degree of reflectance of a probe tone was determined by means of a microphone. Both signal transducer and microphone were directly coupled to the ear. The reflex which produces a change in reflectance is elicited from the contralateral ear. (Technically, the method is apparently similar to that published by J. Zwislocki in *J. Acoust. Soc.* 29:349, 1957 - Rev.).

Sometimes pure-tone audiometry is unable to differentiate neural and transmission losses mostly because of difficulties in masking. In such cases elicitation of the stapedial reflex (or failure to do so) may provide the needed clue. However, the reflex cannot be evoked in some cases, e.g., when the contralateral ear shows a severe transmission loss. As an alternate method the authors investigated the possibility of cutaneous (tactile) elicitation of the reflex from the external canal. Some older reports had hinted at such a possibility.

They finally arrived at a method whereby the cutaneous stimulation was provided electrically. The electrode was built into the ear insert of the measuring device. The authors were able to show summation of the electrically and acoustically elicited reflexes indicating common efferent pathways for both modes of stimulation. There were also apparent differences: The acoustically elicited reflex acts always bilaterally, and lasts for the duration of the applied signal.

The electrically elicited reflex acts unilaterally for weak stimuli and becomes bilateral for stronger stimuli. Response is short lasting. There is adaptation. Several audiological examples are given in which the method of cutaneous elicitation of the stapedial reflex helped to establish a definite diagnosis.

In the discussion of this paper, which was read at the 1959 meeting of the German Otolaryngological Society, A. Thullen made the following remark: These results seem to support a thesis, originally proposed by Gelle. Instead of being elicited via the inner ear, as is the currently accepted theory, the stapedial reflex may always represent a response to tactile stimulation of the eardrum and external canal due to the pressure effect of acoustic signals. Thullen suggested, therefore, to test the feasibility of eliciting the reflex by pressure changes in the external ear canal as in the Gelle test.

Note of the reviewer: The importance of a reliable differential diagnostic procedure in cases of moderately severe hearing losses need not be emphasized, especially with regard to middle ear surgery. On the occasion of a recent European trip, this reviewer was impressed by the widespread use in Scandinavian countries of impedance measurements in clinical audiometry. The consensus of opinion was that, whereas Metz' original method which employs a Schuster bridge, facilitates calibration in terms of absolute units, Anderson's method is easier to apply and, therefore, preferred clinically.

TONNDORF

The Enzymes in Ear Surgery

Antoli-Candela, F.: Acta Oto-Rino-Laringologica Ibero-Americana, Barcelona, 10:4.

The author refers to his experience gained in 450 cases with Chymotrypsin in the following conditions:

- 1) Chronic serous otitis media,
- 2) Chronic adhesive otitis media,
- 3) Transtympanic stapes mobilization,
- 4) Tympanoplasties,
- 5) Faulty epithelialization in mastoid cavities,
- 6) Parenteral administration of the same enzymes in postoperative fenestrations, and ear surgery in which the transtubal installation was also used.

The author believes that in view of the anti-inflammatory proteolytic and fibrolytic properties of the enzyme, the use of this agent diminishes the formation of adhesions and ankylosis, and makes for a cleaner epithelization in tympanoplasties, and fenestrations and stapes mobilizations.

OBREGON

A Contribution to the Rational Treatment of Suppurative Otitis Media

Aragon, Jorge Reyes: Acta Oto-Rino-Laringologica Ibero-Americana, Barcelona, 10:4.

The author made a bacteriological study of 100 patients with acute and chronic suppurative otitis media. These studies were also direct to the sensitivity of the different bacterias against the most commonly used antibiotics.

In his study he found out that 70% of the organisms cultured were sensitive to Chloromycetin, and 65% to Terramycin. The author advises a routine culture and sensitivities studies in all cases of suppurative otitis media, and recommends the Chloromycetin and Terramycin for topical applications in these cases. This, of course, should be followed by daily cleansing of the ear, and in cases where the antibiotics fail, he recommends the use of a solution of 2% Gential Violet. This type of medication is especially very useful in patients who have a great deal of mucoid secretions, and became resistant to antibiotics. This took place in about 20 of the patients studied. The author found out that most of the organisms cultured were resistant to the administration of Penicillin.

OBREGON

Is the Routine Antibiotic Medication of the Acute Suppurative Otitis Media a Justifiable Procedure?

Schroeder, K.: Zschr. Laryng. 39:1, 1960.

This is a unique discussion of the above question, unique because of the author's position: he is the head of a large department of otolaryngology at the medical school at Dresden, East Germany. (This reviewer knows the institution quite well because it was his own father who held that position from 1929 to 1951.) In East Germany antibiotics did not become available generally until quite recently, due

to the postwar development of that country. Even in 1957 practicing physicians used routine antibiotic (preventive) therapy sparingly: 13 out of 20 physicians interviewed as compared to 1 in 20 in 1954. This situation enabled the author to perform a control survey which, although badly needed, nobody in a Western country would dare to make.

All contemporary surveys in the United States, in Great Britain, and in West Germany compare their present figures to those obtained in the pre-antibiotic era, i.e. to the period before World War II. This leaves many questions undecided, for example as to a possible change in the virulence of the bacteria involved.

The author found that in his region of East Germany, even in 1957, the frequency of acute mastoiditis necessitating simple mastoidectomy was quite high: 71% of that in 1938. By contrast, H. House of Los Angeles had told the author that he had not performed a simple mastoidectomy within the last year and a half. Bablik of Vienna reported in 1957 a decline to 11% of the prewar figures. As a whole there had been a steady decline in the frequency of simple mastoidectomies at Dresden since the most recent peak period of 1948, apparently attributable to the gradual increase in the availability of antibiotics. (This reviewer remembers the 1948 epidemic in East Germany quite well from the reports he received from his father. There was no penicillin available in East Germany at that time.) Even in 1948 the incidence in most Western countries was close to zero.

In contrast, cases of acute tonsillitis which had appeared as "dangerous" to the family physician, located in the author's region of the country, had received antibiotics to a large extent (and apparently in sufficient dosages, Rev.). Consequently, the author performed his last ligation of the internal jugular in a case of a tonsillogenic sepsis in 1949. At the same institution, there had been 32 such cases during the period of 1928 to 1948, 20 of which had died as a direct consequence of the disease.

Furthermore, the author mentions several statistics concerning acute suppurative otitis media subsequent to scarlatina, both from Western countries and from East Germany. The Department of Pediatrics at Dresden reported 70 cases of acute otitis media in 584 cases of scarlatina. All of them had received antibiotics early after the onset of the complicating otitis. None of them had developed an acute mastoiditis and consequently no simple mastoidectomies had been performed on these patients.

In comparing his own material to that of Western countries, the author strongly advocates early and routine antibiotic coverage of all cases of acute suppurative otitis media. He stresses the need for otological follow-up of all these patients during the convalescent stage. (There is only one question remaining in the opinion of the reviewer: the discrepancy which might exist in the drug resistance in the East German population as compared to that of Western countries. It appears to be difficult, even from Dr. Schroeder's present material, to answer that question.)

TONNDORF

THROAT

Perforation of the Hypopharynx and Esophagus Due to Foreign Bodies and Instrumentations

Markowicz, A., and Shanon, E.: Otolaryngologica Israeliana 6-7:55-65, 1958.

The authors review the treatment of esophageal perforations and describe in detail the symptoms and signs. It is stressed that following the enthusiasm of the antibiotic era it became slowly obvious that, in spite of this great help, surgical intervention for the purpose of drainage is still important.

During the last 20 years, 26 cases of esophageal and hypopharyngeal perforations were seen in the Ear, Nose and Throat Department of the Beilinson Hospital. Seventeen patients presented themselves with perforations due to foreign bodies. Nine patients suffered a perforation following instrumentation, 1 following bouginage, 1 following oxygen administration under high pressure and 7 following esophagoscopy.

The authors stress the special care that should be taken in esophagoscopy patients with stenosis of the esophagus as well as elderly people where pressing the mucosa with the instrument against prominent or lipped vertebra can be dangerous. Careful postoperative observation should follow apparently uncomplicated endoscopies.

The basic lines of treatment in different stages of inflammation are discussed, keeping in mind always the urgency of the proper treatment. In localized esophagitis the routine includes no intake by mouth, intravenous feeding, antibiotics, esophagoscopy and repeated x-ray studies. If a well-circumscribed abscess is formed it

should be drained. In the case of diffuse infection, bilateral cervical mediastinotomy should be performed. Gastrostomy was found helpful in speeding up recovery.

SADE

Substituting Effect upon the Growth of Thymectomized Guinea Pigs of an Extract Prepared from Human Tonsillar Tissue

Falk, P.: Arch. Ohr., Nas., u. Kehlk.-Heilk. 175:345, 1959.

Guinea pigs when thymectomized during infancy are known to be retarded in body growth. About 60% of such animals die early under the signs of a general cachexia. This effect of an apparent hormonal deficiency can be neutralized by injection of an extract prepared from thymus tissue according to a method developed by Bezsonoff and Comsa. The chemical composition of this extract is not yet known. The author prepared an extract, using Bezsonoff's and Comsa's method, from tissues of human tonsils. When injected with this material thymectomized guinea pigs developed normally. None of a group of 8 animals died during the period covered by the experiment (sacrificed between the 9th and 24th day) as did 50% of the controls (total of 15 animals) which died 17 to 26 days after thymectomy. According to the author, the guinea pig is especially suited for such a substitution experiment since it does not possess any tonsillar tissue.

The author concluded from these results that his tonsillar extract is identical with the thymus extract of Bezsonoff and Comsa. In the discussion of this paper, which was read at the 1959 meeting of the German Otolaryngological Society, S. Rauch, Geneva, Switz., argued that these results may not represent a specific action. According to Rauch, extracts prepared by a different method from salivary glands had shown similar results as those prepared from tonsils. In his closing remarks the author maintained that the thymus-like extract had only been found in a few organs: in about equal amounts in thymus and in tonsils and in minute quantities in lymph nodes and in the spleen. After thymectomy it has vanished from the two latter organs. None of it had been found in salivary glands. Therefore, the effects mentioned by Rauch (which were apparently not demonstrated in thymectomized animals?) are claimed to be of an entirely different nature.

TONNDORF

MISCELLANEOUS

Parasitological Aspects of E.N.T. Practice

Marshak, A.: Otolaryngologica Israeliana 6-7:78-90, 1958.

In many of the arid regions of Israel, open cisterns are used for water reservation. The water is stagnant and breeds leeches and the people and cattle use it at times for drinking purposes, thereby ingesting the parasites. Clinically, the intruder was found to be *Limnatis Nilotica*, which is 3 mm in length before sucking blood and 9 to 10 mm in length and 1 to 5 cm in breadth after sucking blood.

During 24 years of practice in Tiberias, the author has removed 110 leeches from the upper gastrointestinal and respiratory tracts of patients. There were 107 males with only 7 children below the age of 6 years. It is interesting to note that the author never found a leech below the glottis or in the esophagus. The sites of attachment were in the larynx, all parts of the pharynx and the nose.

The symptoms were local bleeding from hirudin secreted by the leeches without any accompanying cough or pain. No swelling was seen at the point of attachment. Children always presented symptoms of stridor which was never seen in adult patients. Removal was performed without difficulty using a Killian polyp forceps through a laryngoscope.

The author saw 27 cases of miasis, only three of which were in adults. In two of the cases, the parasites were found in the nose and in the remaining cases, in the discharging ear.

The ear cases presented symptoms of severe pain while the nose cases not only had severe pain but face swelling and epistaxis. All symptoms subsided promptly on removal of the parasites. The number of larvae found ranged from 1 to 70 and were removed with forceps.

The author saw a 35 year old woman who had bloody sputum, the source of which was found to be *clinostomum compalatum* hooked to her uvula. Although only one other case of this type has been reported, the author points out that in epidemic regions when bloody sputum of unknown origin is encountered, this parasite should be looked for.

SADE

Books Received

Textbook of Otolaryngology

By *David D. DeWeese, M.D.*, Clinical Professor of Otolaryngology, University of Oregon Medical School, Portland, Oregon, and *William H. Saunders, M.D.*, Associate Professor of Otolaryngology, Ohio State University College of Medicine, Columbus, Ohio. Cloth, large 8vo, 464 pages, 354 illustrations. St. Louis, C. V. Mosby Company, 1960. Price \$8.75.

A very useful text, modern in all respects, directed primarily to the medical student and the general practitioner. It covers adequately the details of the specialty required by these readers, and properly de-emphasizes surgical minutiae. References for supplementary study are ample.

What sets this book quite apart from the texts recently turned out is the exceptional excellence of its illustrations. From the simple standpoint of biological photography and illustration they merit special recognition, and the publisher has given them all they deserve in the matter of paper, engraving and presswork.

The Regulation of Cell Metabolism

A Ciba Foundation Symposium, edited by *G. E. W. Wostenholme, O.B.E., M.A., M.B., B.Ch.* and *Cecilia M. O'Connor, B.Sc.* Cloth, 387 pages, 109 illustrations. Boston, Little, Brown and Co., 1959. Price \$9.50.

A highly technical presentation of the subject by sixteen essayists and a number of other authorities participating. Of prime interest to those engaged in basic cell research.

A Doctor's Life of John Keats

By *Walter A. Wells, M.D.* Paper, 8vo, 247 pages, illustrated. New York, Vantage Press, 1959. Price \$3.95.

An engaging book from the pen of our respected old friend and one-time contributor now enjoying retirement from a productive medical career in Washington. Dr. Wells presents us with a sympathetic and analytical picture of the erstwhile student of medicine

turned poet, and dead at twenty-six of tuberculosis and the ineptitude of contemporary medical care. There is appended a short essay on the Mystery of Genius.

Anatomy and Physiology of Speech

By *Harold M. Kaplan, Ph.D.*, Head of the Department of Physiology, Southern Illinois University. Cloth, 8vo, 365 pages, illustrated. New York, McGraw Hill Book Company, Inc., 1960. Price \$8.50.

This is a comprehensive work on the structure and function of the organs involved with the production and reception of speech. It deals with the subject from the standpoint of a physiologist and avoids the clinical and educational problems. Much recent material is included and dealt with in satisfying detail. Its value as a reference book for non-medical people is enhanced by a twenty-four page glossary of terms and phrases.

The Thyroid Hormones

By *Rosalind Pitt-Rivers, F.R.S.*, and *Jamsbed R. Tata*. Cloth, 8vo, 247 pages, tables, and diagrams. New York, Pergamon Press, 1959. Price \$7.50.

This is essentially a compilation of published works on the thyroid, its hormones and their composition, and their effects upon the body tissues. It will interest the otolaryngologist concerned with the underlying causes of the changes in the upper respiratory tract which he has come to associate with the hypothyroid state, although no direct reference is made to specific regions. The bibliography lists 1774 publications, but since the titles are not included it is not possible to say whether any of them deal with the phenomena which the otolaryngologist would like so much to understand.

Diseases of the Nose, Throat and Ear

By *I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E.*, Surgeon to the Royal Infirmary, Edinburgh, Lecturer in Diseases of Nose, Throat and Ear, University of Edinburgh. Cloth, small 8vo, 467 pages, illustrated. Edinburgh and London, E. and S. Livingstone, Ltd. (Williams and Wilkins Co., Baltimore, U. S. Agents), 1959. Price \$5.00.

The sixth revision of this popular handbook since its original appearance in 1937.

Synopsis of Ear, Nose and Throat Diseases

By *Robert E. Ryan, B.S., M.D., M.S. (ALR), F.A.C.S.*, Department of Otolaryngology, St. Louis University School of Medicine, *William C. Thornell, A.B., B.M., M.D.*, Assistant Professor, Department of Otolaryngology, College of Medicine, University of Cincinnati and *Hans von Leden, M.D., F.A.C.S., F.I.C.S.*, Assistant Professor of Otolaryngology, Northwestern University Medical School, Chicago. St. Louis, C. V. Mosby Company. Price \$6.75.

As its title indicates, this is simply a synopsis. On the principle that a little learning can be dangerous, especially in the practice of medicine it would seem that even (or *especially*) the "busy family physician, the intern, and the medical student" to whom it is directed in the preface, would do better to avail themselves of one of the more comprehensive standard textbooks.

La Chirurgie de la Surdit  (The Surgery of Deafness)

By *Prof. M. Aubry*, Clinique-Oto-Rhino-Laryngologique de la Facult  de M decine de Paris, and eight collaborators. Paper, large 8vo, 390 pages, 265 illustrations. Paris, Masson & Co., 1959. Price 5,500 F. (In French).

Das Gutachten des Hals-Nasen-Ohrenarztes (Medicolegal Evidence of Otolaryngologists)

By *Prof. Dr. Med. J. Koch*, Essen, and *Prof. Dr. H. Loebell* M nster/W. Second Edition. Paper, 8vo, 80 pages. Stuttgart, Georg Thieme Verlag, 1959. (Intercontinental Book Corp., N. Y. 16, for U. S. and Canada). Price \$3.20.

Les Maladies du Larynx (Diseases of the Larynx)

By *J. Piquet*, Professeur   la Facult  de M decine de Lille, and *J. Terracol*, Professeur   la Facult  de Montpellier. Cloth, large 8vo, 674 pages, 241 illustrations. Paris, Masson and Co., 1958. Price 9,500 F. (In French).

Il Cortisone e le Sostanze ad azione Cortisonica in Otorinolaringojatria (Cortisone and Its Action in Otorhinolaryngology)

By *Oscar Sala* of the Otorhinolaryngological Clinic of the University of Padua. Cloth, large 8vo, 367 pages, 138 figures. Padua, Minerva Otorinolaringologica, 1959. (In Italian).

Detailed account of experimental and clinical researches. Many photomicrographs, some in color. Forty-one pages of bibliography.

Recuentos Profesionales (Professional Experiences)

By *Dr. L. López Villoria*, Caracas, Venezuela. Laureate thesis for the National Academy of Medicine of Venezuela. Paper, 8vo, 538 pages, 1959. (In Spanish).

Professional recollections of a long life (eighty years at the time of publication), written under the headings of some forty medical subjects.

Die Speicheldrüsen des Menschen (Salivary Glands in Man)

By *Priv.-Doz. Dr. S. Rauch*, Geneva. Cloth, large 8vo, 506 pages, 227 illustrations, some in color. Stuttgart, Georg Thieme Verlag (Intercontinental Book Corp., N. Y. 16, Agents for U. S. and Canada), 1959. (In German).

Thorough-going monograph on the anatomy, physiology and clinical pathology of the human salivary glands with a chapter on clinical diagnosis and another on therapy.

Das Röntgenschichtbild des Ohres (The Tomogram of the Ear)

By *Prof. Dr. K. Mündnich* and *Dr. K.-W. Frey*, Munich. English Translation by *Prof. F. Storer*, London. Large 8vo, cloth, 123 pages, 205 illustrations. Stuttgart, Georg Thieme Verlag, 1959 (Intercontinental Corp., N. Y. 16). Price \$15.70.

Comprises 26 pages of technical data and 93 pages of roentgenological atlas with diagrams. In the usual German *tüchtigkeit*.

Symposium on Pulmonary Ventilation

Ed. by *Dr. R. P. Harbord* and *Prof. R. Woolmer*. Cloth, small 8vo, 109 pages, illustrated. Altrincham, John Sherratt and Son (Williams and Wilkins Co.), 1959.

Symposium (22 participants) held in Leeds on February 19, 1958, under the auspices of the British Journal of Anaesthesia.

Notices

A.M.A. SECTION ON LARYNGOLOGY, OTOTOLOGY AND RHINOLOGY

109th Annual Meeting June 15 - 17, 1960

Miami Beach, Florida

WEDNESDAY, JUNE 15 - 9 A. M.

Joint Session of the Section on Laryngology, Otology and Rhinology,
Section on the Diseases of the Chest, and Section on Radiology

1. Panel Discussion on Tumors of the Trachea and Bronchi

Moderator: Dr. Paul H. Holinger, Chicago 11
Endoscopist: Dr. Herbert W. Schmidt, Rochester, Minn.
Radiologists: Dr. Barton R. Young, Philadelphia
Dr. Wendell G. Scott, St. Louis
Surgeons: Dr. Robert J. Jensik, Chicago 4
Dr. Robert W. Jamplis, Palo Alto, Calif.

2. Panel on Disseminated Diseases of the Chest

Moderator: Dr. Harold O. Peterson, Minneapolis
Internist: Dr. James A. Wier, Denver
Chest Physician: Dr. David Radner, Chicago
Endoscopist: Dr. Walter H. Maloney, Cleveland
Radiologists: Dr. Eugene F. Van Epps, Iowa City
Dr. Clyde A. Stevenson, Spokane
Surgeons: Dr. Donald L. Paulson, Dallas
Dr. William Tuttle, Detroit

THURSDAY, JUNE 16 - 9 A. M.

Audiology: Its Progress and Problems. Kenneth O. Johnson, Ph.D.,
Executive Secretary, American Speech and Hearing Association
(by invitation)

External Otitis: Specific Therapy Based on Accurate Diagnosis. Ed-
ley H. Jones, Vicksburg, Mass.

Address of Guest of Honor: Louis H. Clerf, St. Petersburg

Nasal Tip Surgery. Sam H. Sanders, Memphis

Intermission

Address of Section Chairman: Paul H. Holinger

Problems and Complications in Head and Neck Surgery. George A.
Sisson, Syracuse, N. Y., and Norman E. Johnson

The Long-Term Care of the Laryngectomized Patient. George F.
Reed, Boston

FRIDAY, JUNE 17 - 9 A. M.

Cystadenoma of the Parotid and of the Larynx. Arthur J. Kuhn,
Hammond, Indiana

Effect of Premarin on the Ground Substance. Maurice Schiff, Oak
Knoll, Calif.

Cervico-Facial Congenital Anomalies. Richard T. Farrior, Tampa,
Florida

Incidence of Hearing Impairment in a Medical Center Population.
Edmund P. Fowler, Jr., and Thomas H. Fay, New York

Otometric Operations. George D. Albers, Grand Rapids, Michigan

Intermission

Five Year Results of Stapes Mobilization and Current Results with
Vein Plug Stapedeoplasty. C. M. Kos, Iowa City, Iowa

Fenestration of the Oval Window. John J. Shea, Jr., Memphis

Use of Ear Canal Skin in Myringoplasty. William F. House, Los Angeles

Problems with Tympanoplasty. Edward C. Brandow, Jr., Albany, N. Y.

ROYAL SOCIETY OF MEDICINE, LONDON

The Sections of Laryngology and Otology of the Royal Society of Medicine, London, will be meeting on May 6 and July 14 to 16, 1960. The programs will be as follows:

MAY 6, 10 A.M. *Otology*. Discussion on the Effects of Noise upon Hearing, at which the chief speaker will be Dr. A. Glorig of Los Angeles.

2:30 P.M. *Laryngology*. Papers by Mr. H. D. Brown Kelly on "Dysphagia in Adults (Non-malignant), Its Diagnosis and Management" and by Professor Truls Leegaard (Oslo) on "Management of Chemical Burns of the Esophagus." Mr. Norman Barrett will open the discussion.

JULY. The Sections will hold a combined meeting at Oxford and the program will be as follows:

14TH, 8:30 P.M. Reception by the Presidents of the Sections at Oriel College.

15TH, 9:30 A.M. A paper by Professor Arslan (Padua) on "The Innervation of the Inner Ear" and papers by members of the Otological Research Unit, Medical Research Council, Queen Square, London.

2:30 P.M. Visit to Department of Plastic Surgery, Churchill Hospital.

16TH, 9:30 A.M. Papers by Mr. Desmond Dawes on "The Management of Frontal Sinusitis and Its Complications" and by

Dr. F. H. Kemp and Dr. G. M. Ardran on "The Radiography of the Lower Lateral Food Channels."

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in England at these times will be very welcome. If they wish to come they should write to the Honorary Secretaries of the Sections at 1, Wimpole Street, London, W.1.

<i>Officers</i>	President:	Myles L. Formby, F.R.C.S.	
	Honorary	I. B. Thorburn, F.R.C.S.Ed.	Laryngology
	Secretaries:	E. H. Miles Foxen, F.R.C.S.	
	President:	R. G. Macbeth, F.R.C.S.	
	Honorary	William McKenzie, F.R.C.S.	Otology
	Secretaries:	Charles Gledhill, M.B.E., F.R.C.S.	

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

The next meetings of the American Society of Facial Plastic Surgery will take place on April 6 and July 22, 1960, at the Hotel Elysee, 60 East 54th Street, New York, N.Y., and on October 13, 1960, in Chicago, Illinois.

AMERICAN RHINOLOGIC SOCIETY

An international course in reconstructive surgery of the external nasal pyramid and nasal septum will be given in Mexico City, July 4 - 15, 1960, under the auspices of the University of Mexico Medical School and its Postgraduate Department. This course is given with the co-operation of the American Rhinologic Society, and under the direction of Doctor Andrés Bustamante Gurria, of Mexico City. The guest professor will be Doctor Maurice H. Cottle, Chicago Medical

School, assisted by a faculty of otorhinolaryngologists from the United States and Mexico.

Apply for information to American Rhinologic Society,

Robert M. Hansen, M.D., Secy.

1735 N. Wheeler Ave.

Portland 17, Oregon.

BRITISH COLUMBIA

OTO-OPHTHALMOLOGICAL CONFERENCE

The 1960 British Columbia Oto-Ophthalmological Conference will be held in Vancouver, B. C., on May 11th, 12th, and 13th. Guest speakers will be Doctor LeRoy A. Schall of Boston, in Otolaryngology, and Doctor Charles L. Schepens of Boston, in Ophthalmology.

There will be lectures, round-table discussions, and film sessions, and a social program for the ladies.

Those interested in attending may obtain further information from Doctor G. A. Badger, 925 West Georgia Street, Vancouver 1, B. C.

PAN-PACIFIC SURGICAL ASSOCIATION

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 27 through October 5, 1960.

All members of the profession are eligible to register and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities because of limited space.

Further information may be obtained from Dr. F. J. Pinkerton, Director General, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

THIRD INTERNATIONAL CONFERENCE ON MEDICAL ELECTRONICS

The Electronics and Communications Section of the Institution of Electrical Engineers in association with the International Federation for Medical Electronics, are organizing the Third International Conference on Medical Electronics which will be held at Olympia, London, from July 21 to 27, 1960. Included in the subjects to be covered will be applications to space research, aids for the totally deaf, automatic control of artificial limbs and of heart action, and techniques involving the use of pills containing miniature radio transmitters.

The Conference is designed to bring together members of the medical and electrical engineering professions so that each will gain a better understanding of the problems of the other; besides sessions for experts, there will also be general sessions to enable those who are not at present working in medical electronics to hear surveys of the field, to assess practical applications and to discuss future possibilities.

Further information can be obtained from the Secretary, the Institution of Electrical Engineers, Savoy Place, London, W.C.2.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct only one examination in 1960, and this will be October 3-6, 1960, in Chicago, Illinois, at the Palmer House.

Dean M. Lierle, M.D., Secretary

AMERICAN ACADEMY REGISTRY

The American Academy of Ophthalmology and Otolaryngology has recently established an Employment Registry for Scientists in the

basic sciences pertaining to ophthalmology and otolaryngology. The Registry will aid in the placement of candidates completing training who desire academic and investigative positions and in the placement of those already in such positions who desire different positions. The Registry will also serve institutions who have such positions available.

The Academy will operate this service without fee or obligation, since it feels that the Registry will be of considerable service to many scientists in the fields of ophthalmology and otolaryngology.

Registry forms and further information may be obtained by writing to W. L. Benedict, M.D., Executive Secretary-Treasurer, American Academy of Ophthalmology and Otolaryngology Registry, 15 Second Street S. W., Rochester, Minnesota.

AMERICAN ACADEMY

The 1960-1961 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 15 Second Street S. W., Rochester, Minnesota. Registrations should be completed before August 15.

UNIVERSITY OF BORDEAUX

A practical graduate course in surgical techniques, Otolological Functional Micro-Surgery, will be given in English from June 26th to July 5th, 1960, by Michel Portmann, M.D. (Associated professor).

The number of students is limited to 20, divided into two groups. Fees, \$100 (to be paid at the Faculty of Medicine in Bordeaux at the beginning of the course).

Applications should be sent to Docteur Michel Portmann, 45, cours Foch, Bordeaux.

CASSELBERRY PRIZE

A sufficient fund having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available in part or as a whole, for a prize award. Theses must be in the hands of the Secretary of the American Laryngological Association prior to January 1 of any given year.

The Award is a prize of money with accompanying certificate signed by the officers of the American Laryngological Association. The sum of money will be agreed upon by the Council of the Association after the manuscript has been evaluated by the Award Committee. It may be awarded in whole or in part among several contestants.

Eligible contestants may be: 1) hospital interns, residents, or graduate students in Rhinology and Laryngology; 2) an individual with an M.D. degree who is actively practicing or teaching Rhinology and Laryngology in the Americas; 3) any scientific worker in the field of Rhinology and Laryngology.

Manuscripts shall be presented to the Secretary of the Association under nom de plume which shall in no way indicate the author's identity. There shall also be a sealed envelope bearing the nom de plume and containing a card showing the name and address of the contestant which the Secretary shall keep in his possession.

Manuscripts must be limited to 5000 words and must be type-written in double spacing on one side of the sheet. The thesis shall not have been published elsewhere before submission.

The successful thesis shall become the property of the American Laryngological Association but this provision shall in no way interfere with publication of the thesis in the journal of the author's choice. Unsuccessful contributions will be returned promptly to their authors.

The Award which will be made at the Annual Meeting of the American Laryngological Association shall be based on:

1. Originality of material
2. Scientific and clinical value

3. Suitability for this Award
4. Method of presentation as to style, illustrations and references.

The maximum amount of the Award shall not exceed \$200.00.

Lyman G. Richards, M.D., Secretary

MOUNT SINAI HOSPITAL

An intensive postgraduate course in rhinoplasty, reconstructive surgery of the nasal septum and otoplasty will be given July 16, 1960, through July 29, 1960, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

TEMPLE UNIVERSITY

There will be given a postgraduate course in Laryngology and Laryngeal Surgery April 4th to 15th and September 26th to October 7th, 1960, and a postgraduate course in Broncho-esophagology February 1st to 12th and November 7th to 18th, 1960 in the Department of Laryngology and Broncho-esophagology, Temple University Medical Center, under the direction of Drs. Chevalier L. Jackson and Charles M. Norris.

The tuition fee for each course is \$250. Information may be obtained by writing to: Jackson-Research, Lab 604, Temple University Medical School, 3400 N. Broad Street, Philadelphia 40, Pa.

UNIVERSITY OF ILLINOIS

The next postgraduate course in Laryngology and Broncho-esophagology to be given by the University of Illinois College of Medicine is scheduled for the period April 4 to 16, 1960. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

THE GOULD AWARD

The International Committee for the Gould Award announces the selection of the recipient for the year 1959—Professor Joel J. Pressman, M.D., University of California at Los Angeles—for his anatomic and radio-isotope studies of laryngeal compartments.

The Gould Award is presented annually for basic research in laryngology to encourage and reward fundamental investigations in this field.

ANNALS

In order to fulfill the requests for the March 1959 and June 1959 issues, the ANNALS will pay \$3.50 for each book in good condition.

Please mail books to Zimmerman-Petty Linotyping Co., 2308 Olive, St. Louis 3, Mo.

OFFICERS

OF THE

NATIONAL AND INTERNATIONAL OTOLARYNGOLOGICAL SOCIETIES

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: John H. Dunnington, M.D., New York

Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

Meeting: Palmer House, Chicago, October, 1960.

AMERICAN BOARD OF OTOLARYNGOLOGY

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AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

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